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A  
HAND-BOOK  
ON THE  
DISEASES OF THE HEART,  
AND THEIR  
HOMŒOPATHIC TREATMENT.

BY

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*O-School of Medicine*

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TO  
THE ABLE  
PHYSICIAN,  
MY MUCH ESTEEMED  
FRIEND AND WHILOM PRECEPTOR,  
G. W. BARNES, M. D., OF SAN DIEGO, CAL.,  
THIS BOOK IS MOST  
RESPECTFULLY  
DEDICATED.



## PREFACE.

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This little book had its beginning in a Course of Lectures on the subject delivered by the Author in a Medical College some years ago. In its preparation, the endeavor has been to produce a work which, while not too voluminous, should contain in a convenient form, all that is most reliable and useful of the known facts in relation to this important subject, and thus be up to date in both pathology and therapeutics. That it is necessarily imperfect in many respects, no one can be more fully aware than the author.

Although he has drawn largely from his own observation and experience, which in this class of affections at least have been quite extensive, yet for many points in physiology and pathology, he is especially indebted to the writings of Stokes, Flint, Fothergill, Hayden, Raue, Vogel, Lebert, Rosenstein, Bauer, Schroetter,



and many others, and in *materia medica* and therapeutics, to our own standard authors.

Hitherto, far too little attention has been given to the diseases to which the heart is subject, whether with respect to pathology, diagnosis, or treatment, and it is much to be regretted that there are so few physicians of any school, who can accurately diagnose even the simplest form of disease of that organ; and if this book proves to be the means of inducing any physician to study more closely this class of diseases, or causes one poor mortal to enjoy a few more years of life and health than he otherwise would have done, it will not have been written wholly in vain.

W. P. A.

# CONTENTS.

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## CHAPTER I.

	PAGE.
ANATOMY AND PHYSIOLOGY OF THE HEART, . . .	17

## CHAPTER II.

PHYSICAL EXAMINATION OF THE HEART, . . .	27
Physical signs of the heart in Health—Inspection, Palpation, Percussion, Auscultation. The sphygmograph.	

## CHAPTER III.

PHYSICAL SIGNS IN CARDIAC DISEASES, . . .	37
Pericardial Adhesion, Bulging, Displacement of Apex, Resistance, The Pulse, Intermittency, Irregularity, Tumultuous action, Increased dullness, Exaggeration and Suppression of Heart Sounds, Murmurs, Friction sounds.	

## CHAPTER IV.

EFFECTS OF OBSTRUCTED CIRCULATION, . . .	51
--	----

## CHAPTER V.

PERICARDITIS, . . . . .	58
Physical signs, Percussion, Auscultation, etc., Terminations, Diagnosis, Prognosis, Treatment.	

	PAGE.
HYDROPERICARDIUM, . . . . .	83
Symptoms, Diagnosis, Prognosis, Treatment.	
PNEUMOPERICARDIUM, . . . . .	86
Physical signs, Treatment.	

## CHAPTER VI.

DISEASES OF THE WALLS OF THE HEART, . . . . .	88
HYPERTROPHY.	
Etiology, (Obstruction, Increased pressure, Habitual excitement.) Symptoms, Physical signs and consequences.	
DILATATION, . . . . .	100
Primary and Consecutive ; Physical signs, Prognosis, Treatment.	
FALSE HYPERTROPHY, . . . . .	127
Etiology and Treatment.	
CARDIAC ATROPHY, . . . . .	128
Etiology, Diagnosis, Prognosis and Treatment,	
MYOCARDITIS, . . . . .	131
Causes, Diagnosis, Prognosis, and Treatment.	
FATTY DEGENERATION, . . . . .	134
Conditions, Causes, Physical signs, Prognosis, Treatment.	
FATTY INFILTRATION, . . . . .	153
Physical signs, Diagnosis and Treatment.	
CARDIAC SOFTENING, . . . . .	156
Simple softening, Physical signs, Treatment.	

## CHAPTER VII.

CANCER AND WOUNDS OF THE HEART, . . . . .	158
Syphilitic nodules, Hydatids, Tubercles.	
ANEURISM, . . . . .	159
Physical signs, Diagnosis, and Treatment.	



## CONTENTS.

13

	PAGE.
WOUNDS OF THE HEART, . . . . .	161
Diagnosis and Treatment.	

### CHAPTER VIII.

ENDOCARDITIS, . . . . .	163
Acute and Chronic, Causes, Physical Signs, Diagnosis, Prognosis, Treatment.	

### CHAPTER IX.

VALVULAR DISEASES, . . . . .	179
<i>Mitral Obstruction</i> , . . . . .	181
<i>Mitral Insufficiency</i> , . . . . .	184
<i>Aortic Valvular Disease</i> , . . . . .	189
<i>Aortic Insufficiency</i> , . . . . .	197
<i>Tricuspid Regurgitation</i> , . . . . .	198
<i>Tricuspid Obstruction</i> , . . . . .	199
<i>Pulmonic Regurgitation</i> , . . . . .	200
Treatment of the various forms.	

### CHAPTER X.

EMBOLISM, . . . . .	209
Causes, Signs, and Treatment.	

### CHAPTER XI.

NERVOUS PALPITATION, . . . . .	209
Causes, Physical signs, Treatment.	

### CHAPTER XII.

IRRITABLE HEART, . . . . .	216
Causes, Signs, Treatment.	

## CHAPTER XIII.

GRAVE'S OR BASEDOW'S DISEASE—EXOPHTHALMIC GOITRE, . . . . .	219
---	-----

## CHAPTER XIV.

ANGINA PECTORIS, . . . . .	225
Homœopathic Treatment.	

## CHAPTER XV.

DISEASES OF THE AORTA—ATHEROMA, . . . . .	236
---	-----

## CHAPTER XVI.

THORACIC ANEURISM, . . . . .	240
Etiology, Diagnosis, Treatment.	

## CHAPTER XVII.

CONGENITAL DEFECTS, . . . . .	249
Stenosis of Pulmonary Artery, Stenosis of Foramen ovale, Interventricular Septum Ventricles, Diagnosis, Terminations.	

## CHAPTER XVIII.

CAUSES OF CARDIAC DISEASES, . . . . .	256
Distinction between Cardiac and Non-Cardiac Diseases.	
Elements of Prognosis.	
INDEX, . . . . .	269



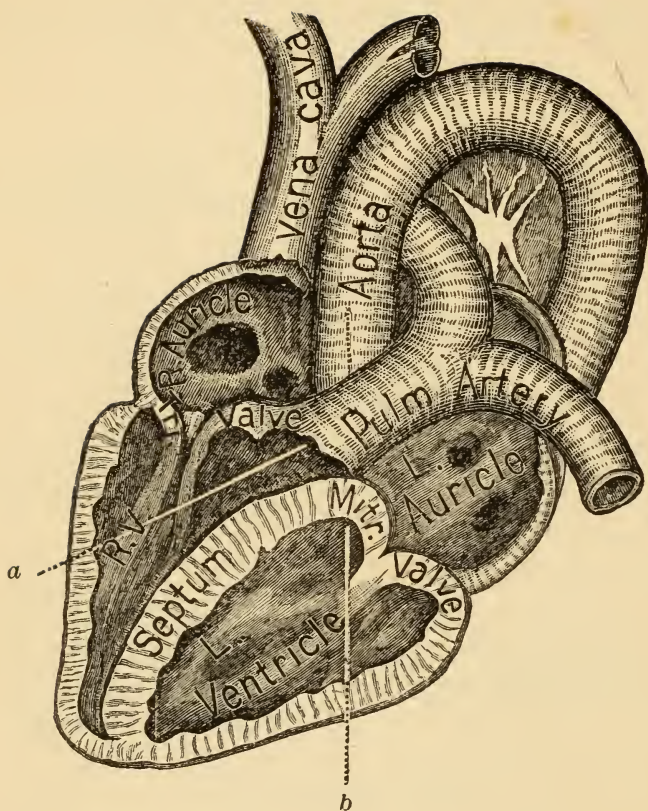


FIG. II.

Vertical section of Heart, as seen from the left, showing its peculiar anatomy. *a*. Line passing through into pulmonary artery, showing the direction the blood takes on leaving the right heart. *b*. Line showing the exit of blood from the left heart through the aorta.

# THE HEART AND ITS DISEASES.

## CHAPTER I.

### PHYSIOLOGICAL ANATOMY OF THE HEART.

The heart is a hollow, involuntary muscle, containing four cavities: a right auricle, a right ventricle, a left auricle and a left ventricle. It is enveloped in a fibro-serous membrane called the pericardium, and is situated with its centre a little to the left of the mesian line of the thorax, about one-third of it being to the right, and two-thirds to the left of that line. Its apex points downwards, forwards and to the left, as far as the fifth intercostal space, and about an inch to the right of a line running vertically through the left nipple while its base points upwards, backwards and to the right, as far as the upper border of the third right costal cartilage. To the right it extends from half an inch to an inch to the right of the sternum; to the left it extends almost, and frequently quite to the mammillary line, while its lower border rests upon the diaphragm. Its base is attached by the great vessels to the fibrous layer of the pericardium, its apex being entirely free.



The heart is the great centre and chief propelling power of the circulation. The impure blood coming into the right auricle from the superior and inferior venæ cavæ, passes into the right ventricle, from which it is prevented from returning by the tricuspid valve; from the right ventricle it passes, at each ventricular systole or contraction, into the pulmonary artery, its return being prevented by the semi-lunar valves at the mouth of that vessel. The pulmonary artery conveys the blood to the lungs, where it is purified by contact with the air to be returned as arterial blood through the four pulmonary veins, to the left auricle, from which it passes into the left ventricle. From this it is prevented from returning by the mitral valve. At each contraction of the left ventricle, the blood it contains is emptied into the aorta, the aortic semi-lunar valves guarding against its return. Through the aorta and its branches it is carried to all parts of the system, and after passing through the capillaries, it returns through the veins to the right auricle as before.

The auricles constitute the base of the heart, and the ventricles the apex, the line of separation between the two portions being marked exteriorly by a transverse depression, the auriculo-ventricular sulcus. As the ventricles are called upon to exert a greater amount of strength than the auricles, their walls are thicker, while those of the left ventricle are considerably thicker than those of the right, as it has a much harder task imposed upon it, viz: to throw the blood to the capillaries of the entire system. As different hearts



have walls of varying thickness, (and different authors fail to agree even as to the relative thickness of different parts) it would be impossible to fix upon any definite thickness for the walls of each part in health. Flint estimates that of the right auricle at one twelfth of an inch, that of the left somewhat greater, that of the right ventricle at about one sixth, and of the left, at nearly half an inch; the relative thickness of the two ventricles being as about one to three. Gray says in his *Anatomy*, one to two. The heart walls in the female are thinner and more delicate than those of the male.

The size of the heart varies, its dimensions depending largely on whether it is full or empty, expanded or contracted; it may be safe to say, however, that it measures about four and a half inches in length from base to apex, and about four inches in breadth, and that it is slightly flattened from before backwards. Gray's measurement must have been taken from the heart when it was entirely empty and its walls contracted, as he makes the length five inches the breadth three and a half, and the antero-posterior diameter only two and a half inches; but this is not a fair measurement. The average weight is from eight to ten ounces. Craveilhier says seven to eight ounces, Hayden nine to ten, and Marshall eight to twelve ounces.

Having considered the heart as a whole, we now proceed to a consideration of its individual parts, only touching, however, upon those more important points

which will be of most use to us in treating of the several abnormal alterations and conditions to which it is subject.

The right auricle has three large openings, the mouths of the superior and inferior venæ cavæ, and the right auriculo-ventricular opening, or passage from the right auricle to the right ventricle. There are also the mouths of the coronary vein, and the minute veins known as venæ Thebesii, all of which convey the venous blood from the heart walls into the auricle, and open obliquely; so that when it contracts, they close and prevent the blood from regurgitating and filling the heart walls with de-oxygenized blood just at the moment when its greatest strength is required. The auricular appendages are two hollow dentated projections, on the anterior portion of the auricles. Their inner surface is marked by comb-like muscular columns called *musculi pectinati*.

In the right ventricle we have the auriculo-ventricular opening, the opening of the pulmonary artery, the *conus arteriosus*, the tricuspid valve, the semi-lunar valves, the *chordæ tendineæ*, and the *columnæ carneæ*.

The auriculo-ventricular opening is about an inch in diameter, is surrounded by a fibrous ring, and guarded by the tricuspid valve. It is situated at the base of the ventricle.

The *conus arteriosus* is a conical extension on the left anterior and superior portion of the right ventricle, from which the pulmonary artery takes its origin.

The mouth of this artery is guarded by the semi-lunar valves.

The tricuspid valve consists of three triangular segments or curtains, mostly of fibrous tissue, which close the orifice by their free edges coming in contact on being pressed backward by the blood during ventricular contraction.

The semi-lunar valves are three in number, and close the pulmonary artery on being pressed backward by the return current during diastole.

The left auricle has five openings, the mouths of the four pulmonary veins, and the auriculo-ventricular opening. It has also, on the internal surface of its appendix, a few muscoli pectinati, which are smaller than those of the right side.

The auriculo-ventricular opening is smaller than that on the right side, and the fibrous ring which surrounds it is firmer.

The left venticle has for consideration, the auriculo-ventricular and aortic openings, the mitral and semi-lunar valves, the columnæ carneæ, and the chordæ tendineæ.

The aortic opening is small and circular, and is guarded by the semi-lunar valves, which are three in number as on the right side. It is situated to the right, partly anterior, and in very close proximity to the left auriculo-ventricular opening. The mitral valve, which guards the latter orifice, is firmer and stronger than the tricuspid, and consists of two segments, one of which is larger than the other.

The segments of both the tricuspid and mitral valves are somewhat thicker in their centres, to which are attached several strong tendinous cords, the chordæ tendineæ. Some of these cords spring directly from the ventricular walls, while others take their origin from certain of the fleshy columns.

The columnæ carneæ or fleshy columns, found projecting from the walls of both ventricles, are smaller and more numerous in the right than in the left. They are of three kinds: those which are attached by their whole length to the heart wall, those which are attached at both extremities and free in their centres, and those which are attached by one extremity only, the other being connected, by the tendinous cords, to the valve segments. The last mentioned variety are called muscoli papillares, and each sends tendinous cords from the same base to different valve segments, so that when these muscles contract with the contraction of the ventricle, they have a tendency to draw the flaps together, and thus close the orifice. A further use of these chordæ tendineæ, a use not only of those which originate in the papillary muscles, but those which spring from the fixed wall or inter-ventricular septum, is to prevent the valve segments from being washed backwards into the auricle during ventricular contraction, which would permit regurgitation.

The semi-lunar valves in both the aorta and pulmonary artery, have peculiarities common to both. Both sets are three in number; both have thin free borders called lunulæ, and the summit of each seg-



ment in both is crowned by a hard fibrous tubercle, called corpus Arantii. These tubercles and free borders come into direct contact during cardiac expansion or diastole, thus effectually closing the respective orifices and preventing regurgitation.

Behind the semi-lunar valves in both vessels, are certain depressions in the walls, which serve as recesses for the valve segments during ventricular contraction, and these recesses are called sinuses of Valsalva. The valve segments, the lunulæ, the corpora Arantii, and the sinuses of Valsalva are all smaller, less perfect, and less distinct at the pulmonary than at the aortic orifice.

The endo-cardium is the serous membrane which lines the internal surface of the heart. It covers its walls, its tendinous cords, and its columns; is duplicated upon and adds strength to its valves, and is continuous with and similar in structure to the lining membrane of the great blood vessels. It is thinnest in the ventricles and transparent, thickest in the auricles, especially the left, where it is somewhat opaque and white.

The pericardium is composed of two layers, one serous and the other fibrous. The fibrous layer is strong and dense, and is attached above to the great vessels and by means of these and the cervical fascia to the upper ribs and clavicles, to the sternum in front by the anterior mediastinum, below to the diaphragm, and posteriorly by the posterior mediastinum to the spinal column. The serous layer invests the heart, and is then duplicated upon the internal surface of the

fibrous layer. Like all serous membranes, it is smooth and shining, and secretes in health a small quantity of thin fluid which facilitates the movements of the contained organ.

But how does the heart, being, as it is, the great central propelling organ of the circulation, sending at each contraction of its ventricles a fresh supply of pure blood to every part, receive in its turn the blood which it requires for its own nourishment? It has been supposed by some, and I believe is still claimed by one author at least, that this is effected in a direct manner by the absorption of blood from its own cavities; that the many little prominences, folds, and lacunæ, which line its inner surface, contribute to this end; that it thus receives the first and richest of the blood for its own use; and that the coronary arteries are veins with reference to it, emptying venous blood into the aorta instead of taking arterial blood from it for the heart's supply. This theory is, however, disproved by certain well known facts in anatomy and physiology. The entire internal surface of the heart, with all its prominences and all its indentations, is lined by a double membrane, a fibrous and a serous layer, and on the left side, which contains the pure blood, there are in this membrane no foramina, the mouths of vessels extending into the heart substance, the most careful examination having failed to reveal them, so that the walls could not possibly receive their blood supply by such means. Again, serous membranes are only to a very limited extent absorbing surfaces,



and it is not probable that enough blood for the purpose could be absorbed at each diastole by the small surface contained in the cavities of the left side; besides, the absorbing surface would not be likely to take up whole blood corpuscles ready for use. Again, that the heart should take its blood from its own cavities for the sake of receiving the first and purest is altogether unnecessary, as that poured into the aorta from the left ventricle is equally pure.

The commonly received and more rational theory is this. Branching off from the aorta, behind the semi-lunar valves, one in each of the two anterior sinuses of Valsalva, are the coronary arteries, which are distributed to the heart walls. These receive their supply of blood from the aorta during diastole, or the period of ventricular relaxation and expansion, the propelling force *not* being the cardiac contraction by means of which the other arteries are filled, but the *aortic recoil*. The aorta, being filled and distended by the ventricular contraction, recoils by its own elastic nature, and, forcing a portion of the blood backward against the semi-lunar valves, closes them and fills the coronary arteries during diastole, so that at the time when most needed, that is, during systole, or contraction, the heart, has just received and appropriated a fresh supply of blood. This blood passes on through the cardiac capillaries, and, during diastole, is emptied as venous blood, through the coronary vein and *venæ Thebesii*, into the right auricle.

Hayden, in his recent work upon the heart, main-

tains that a large portion of the heart's blood supply enters the coronary arteries from the aorta during systole, but this is evidently impossible, from the contracted condition of the walls at that time, if for no other reason.

The heart is an organ having the property of contraction and relaxation at regular intervals, performing all its movements in regular cycles of time, and this power is independent of the will, and retained even after the heart has been entirely severed from the body. This organ, after being cut out of the living subject, will, upon being irritated upon its inner surface, alternately contract and dilate with regularity, and in certain cold-blooded animals, this irritability continues for many hours; in some, the heart continuing to pulsate regularly for a long time after its connection with the body has been severed; and this it does even without any irritation.

This involuntary muscle, then, has a nervous system of its own, and this nervous system consists of ganglia imbedded in its substance, mostly about the auriculo-ventricular sulcus. These ganglia are no doubt aided very materially by their connection with the spinal cord, but especially with the deep and superficial cervical ganglia. Upon this ganglionic nervous system depends that irritability which causes the heart to contract when its cavities are filled with blood. This nervous force is regulated and held in check by the inhibitory nerves, the cardiac branches of the pneumogastric.

Another important part in the cardiac innervation, is that of its bloodvessels. These derive their contractility from the vaso-motor nerves, which are counteracted and held in check by the vaso-inhibitory nerves. If the vaso-motor nerves are excited to increased action, the coronary vessels contract so as to produce anæmia of the heart-walls. On the other hand, if the vaso-inhibitory nerves are unnaturally active, those vessels are dilated instead, and we have hyperæmia.

If the pneumo-gastric nerve be irritated at its origin, it exerts in an increased degree its effect upon the heart, which ceases to pulsate, and remains in diastole. On the other hand, if the cervical portion of the spinal cord be irritated, the heart's activity is increased.

But this is not all; the nerves above mentioned do not complete the cardiac nervous system, as it is rendered more complex by the addition of other branches whose functions are not definitely known.

In order to propel the blood to all parts of the system, the heart must, at each contraction, exert an immense force, and as it expends this force over four thousand times every hour, night and day, as long as life lasts, it becomes necessary that it should have some rest, and this it finds after every pulsation. During the stage of partial expansion, while its cavities are gradually filling, after its vessels have been well supplied with pure blood from the aorta, it remains in a passive state, and thus takes its momentary rest,

to resume its work when its ganglionic nervous system, feeling the pressure of the blood, tells it that its cavities are filled and it must again contract.

By some authors this rest is called sleep, and Fothergill\* thinks that nineteen hours in every twenty-four are thus spent, but I cannot agree with him in this. It may be that it rests in this manner one-half of the time, certainly not much more, and during periods of cardiac excitement from whatever cause, the time thus spent is much less, the increased frequency being not at the expense of both the pulsation and pause, but of the pause almost or entirely alone, so that the time between the second and first sound is often no longer than that from the first to the second.

\* The Heart and its Diseases.



## CHAPTER II.

### PHYSICAL EXAMINATION OF THE HEART, AND PHYSICAL SIGNS IN HEALTH.

No perfectly reliable diagnosis of a heart affection can be made without a physical examination; and as no medicine can be intelligently prescribed in disease without first knowing its effect when administered to the healthy, it is equally necessary, in order to make an intelligent physical examination of a diseased organ, that we first know the signs which would be discovered by a physical examination of the same organ in health. It is such an examination which we now propose briefly to consider. It consists of the following four principal parts:

1. Inspection; examination by the eye alone.
2. Palpation; examination by the touch.
3. Percussion; examination by tapping upon the region to be examined, either directly, or, which is preferable, upon one or more fingers applied to the surface and listening to the sounds thus produced.
4. Auscultation; examination either by the ear applied directly to the chest walls, or through the stethoscope. While immediate auscultation is better in pulmonary affections, I would most earnestly recommend that no thorough examination of a case of

chronic heart disease be attempted without a good stethoscope; and this should not be the ordinary wooden tube, but a bin-aural one, one that has no wire springs to loosen and rattle, and thus interfere with the transmission of the unadulterated heart sounds. Great care should be taken in selecting the instrument, to find one which fits the ears perfectly, as, if too large or too small, there will be much roaring connected with its use, a very serious defect.

Before proceeding to examine the patient, the whole anterior portion of the chest should be divested of all covering, and in some cases the posterior portion also, for nothing should come between the stethoscope and the skin. Without this preparation, not only are palpation, percussion and auscultation greatly interfered with, but inspection is usually rendered entirely valueless. My usual practice is to make the greater part of the examination while the patient is sitting erect, only *varying* the position by having him lie down upon the back. The room should always be comfortably warm.

If, the patient being now ready, the physician remembers this one important point in regional anatomy, that the rudimentary nipple is usually found about the lower border of the fourth rib, it will often save him much embarrassment.



## PHYSICAL SIGNS OF THE HEART IN HEALTH.

*Inspection.*—If we examine the heart of a healthy person in the erect position, the apex will be found beating in the fifth intercostal space, about an inch to the right of a line running vertically through the left nipple. If the subject be somewhat thin in flesh, the impulse will appear more diffused, but if it be a woman in good flesh and with largely developed mammæ, the impression made by the apex upon the thoracic parietes will be but slightly if at all perceptible to the eye. If now the person lie down upon the back, the apex may in many cases be seen to rise, sometimes appearing as high as the fourth intercostal space, while if he turn upon the left side it will move to the left to the extent of nearly an inch, or if upon the right side, it will fall about half as far to that side, thus always seeking the lowest point; for the apex is free in the pericardium, in which it lies as in a bag, which closely envelops it, follows its movements, and, to a considerable extent, retains it in its proper position. In the course of this work, when speaking of the situation of the apex, it will generally be assumed that the patient is sitting erect, and allowance must be made for any deviation which will of necessity take place in its position in the different attitudes which he or she is likely to assume.

*Palpation.*—Unless the mammary development is great, or there is some deformity, the apex can nearly

always be felt in the healthy person, beating in its proper position.

*Percussion* displays two kinds of dullness; superficial and deep. Superficial dullness is that which is displayed on slight percussion. It marks that portion of the heart which lies next the anterior chest wall, and is limited to an irregularly triangular space having for its base the middle line of the sternum, for its superior angle the centre of the sternum opposite the fifth costal cartilage, for its inferior angle the centre of the sternum opposite the sixth costal cartilage, and for its apex a point about half an inch or an inch above and to the right of the apex of the heart, its inferior border being rounded slightly downwards, while the upper border is nearly straight. This area is about two inches in diameter from above downwards at its widest point, and about three inches from base to apex.

Deep dullness is displayed on percussion with a much firmer pressure, and marks that portion of the organ which is covered by the thin edges of the lungs projecting between it and the chest walls. Its outlines, therefore, are the outlines of the heart and beginning of the great vessels above. This is frequently called the *deep precordial region*, and, like the region of superficial dullness, is also irregularly triangular in shape, its rounded apex, about two inches in width, being above, corresponding to the beginning of the pulmonary artery, the ascending aorta and anterior portion of the arch, and extending obliquely across

the sternum from the upper edge of the second left costo-sternal articulation to the lower edge of the corresponding articulation on the right side. The right hand border corresponds to a line drawn vertically downwards from the last named point to the upper edge of the sixth costal cartilage, is situated a little to the right of the sternum, and is rounded slightly outwards. The base of this triangle is below, is marked by the diaphragm, is about four inches in length, and corresponds to a line drawn from the inferior extremity of the right hand border just described, to the apex of the heart in the fifth intercostal space. The left boundary is rounded slightly outwards, and extends from the apex of the region we are now describing at the superior border of the second costal cartilage of that side, about half an inch from the sternum, to the apex of the heart in the fifth intercostal space, a little inside the nipple line.

The dullness observed in this region is not absolute as in the superficial precordial region, but is only partial, and during a full inspiration is almost or quite imperceptible.

The difficulties in the way of the diagnosis of heart affections by percussion, are many and of frequent occurrence. Among them may be mentioned effusions into the pleural sac, which throw the heart out of its proper position; pulmonary emphysema which diminishes the region of superficial dullness and adds to the resonance of the deep precordial region; pleuritic adhesions, drawing the lung down over the heart; infl-



trations into the substance of the lung, creating dullness over the part affected, which, if near the heart, will be likely to give rise to confusion as to the limits of cardiac dullness; thoracic aneurisms; deformities of the chest walls, whether congenital or acquired; largely developed mammæ, or excessive adipose.

*Auscultation* betrays two sounds, a first and a second; there being, from the second sound of one pulsation to the first sound of the next pulsation, a considerable pause or rest, embracing a little more than half the time from the beginning of one pulsation to the beginning of the next, so that, as in the adult, the duration of the entire pulsation is not quite one second, that of the pause may be set down at a little more than half a second. This period of quiescence is called the long pause, while the interval from the first to the second sound is called the short pause.

The first sound is systolic, or synchronous with the contraction of the ventricles, and is composed of two elements, one of which is muscular and produced by muscular action and the sudden pressure of the apex against the anterior chest wall, and giving that heavy sound characteristic of it; the other valvular, and caused by the sudden tension at the mitral valve as it is closed by the pressure of the blood in the ventricle, the tricuspid sound being so faint as scarcely to be heard.

The second sound is diastolic, occurring at the moment when the ventricles begin to expand, and is produced by the closing of the valves of the aorta and

pulmonary artery, as the recoil of the elastic coats of those vessels suddenly forces the blood back upon them.

The first sound is heard most distinctly about the apex of the heart, while the greatest intensity of the second sound is over the semi-lunar valves in their respective situations, and over the beginning of the great vessels at the mouths of which they are situated, the pulmonary sound being heard on the left side, in the second intercostal space, at the upper margin of the third rib, close to the sternum, and the aortic sound in the second intercostal space, on the right side, close to the sternum. These sounds are also heard at other places, generally all over the precordial region, but their maximum intensity is at the points which I have mentioned. Hayden claims that sounds produced at the aortic opening are heard loudest in the middle of the sternum.

*The Sphygmograph.*—It may be thought by some, that in a work upon the diseases of the heart, more attention should be given to this clever invention than I have seen fit to bestow upon it. I omit its tracings for two reasons. The first is, that this book is intended as a hand-book containing practical knowledge for every day use, and it is not supposed that very many of those who will read it are possessed of such an instrument. The second reason is, that as far as they have been yet developed, sphygmographic tracings are of but little practical value as a means of diagnosis, not one of them being pathognomonic of any par-

ticular lesion. The nearest approaches to a pathognomonic character possessed by any of them, are found in aortic regurgitation, and narrowing of the aortic orifice, when either of these exists without any complication, yet even these are sometimes closely simulated by tracings in other conditions.

The sphygmograph certainly does show the degree of arterial tension, and hence, by repeated use in a given case, may be made to demonstrate a progressive heart failure or an improvement in tone of the ventricular walls, as the case may be. In this respect it might be of much practical value to a physician who has been deprived of any of his faculties, especially the sense of touch, but otherwise it could be of but little use.



## CHAPTER III.

### PHYSICAL SIGNS IN CARDIAC DISEASES.

Before treating of the several diseases to which the heart is subject, it may not be amiss to detail some of their physical signs.

A *systolic retraction* of the lower portion of the sternum and the left inferior costal cartilage, followed by a *diastolic recoil* of the same parts, is pathognomonic of pericardial adhesion. This retraction is most likely produced by the energetic action of a heart which, through the pericardium, is adherent by its inferior surface to the diaphragm.

Undue *prominence* of the precordial region may be due either to enlargement of the heart itself, or to a considerable pericardial effusion. *Bulging* of the intercostal spaces in the precordium, would be indicative of quite extensive pericardial effusion.

The apex may be *displaced* to the *left* of its normal position, generally indicating some form of cardiac enlargement, usually the result of valvular disease; such displacement is also found when the heart is either misplaced to the left, or when an elevated diaphragm has caused it to assume a horizontal position. If the impulse of the apex is displaced *downward* as well as to the left, it is an indication that the enlarge-

ment is considerable. It is sometimes displaced far to the *right*. It then indicates extensive pleuritic effusion of the left side. Again, the impulse may *not be perceptible* either to the eye or the touch. When this is not the result of the presence of large mammæ, excessive adipose, or pulmonary emphysema, it is indicative either of great cardiac debility, as in atrophy or fatty degeneration, or of extensive pericardial effusion.

*Pulsation* in the right first and second intercostal spaces, close to the sternum, is indicative of dilatation of the aorta at that point. If found in the second intercostal space alone, in a very thin subject, it possesses no significance. In either case it is systolic in time.

*Pulsation* in the *jugular veins*, generally both visible and palpable, is indicative of tricuspid regurgitation with consequent giving way of the venous valves. It is generally accompanied by *pulsation* in the *liver*.

In persons possessed of an equal degree of fatty or mammary development, the heart conveys to the hand the impression of greater or less *resistance*, from the powerful impulse met with in the pure hypertrophy consequent on narrowing of the aortic orifice, down through the different degrees of dilatation and debility until it is lost to the sense of touch in the advanced stage of fatty degeneration.

The so-called "*cat's purr* or *fremissement cataire*," is a sensation like that felt when placing the hand on a cat's back while purring. It is supposed to be produced by eddies in the blood current. As an endocar-

dial symptom, it is most frequently felt over the left ventricle, immediately before the first sound and impulse, and is then indicative of narrowing of the mitral orifice. It is also met with in the veins in cases of anæmia. It is different only in degree from the *grating* sensation felt in pericarditis, as the result of the rubbing together of the two inflamed surfaces of the pericardium, and known as tactile fremitus. The vibration of pericarditis does not commonly amount to genuine *fremissement cataire*.

*Hardness, loss of elasticity, and tortuousness of the arteries* are generally due to inflammation of their coats. It is frequently indicative of disease of the aortic valves.

Aside from this want of elasticity in the arteries, the *pulse* often possesses characteristics of great value in diagnosis. Thus it may be either *irregular* or *intermittent* or *both*. If these symptoms show themselves worse after exertion, they are indicative of heart failure.

A pulse which is somewhat *smaller* than natural, but *slow, firm, and prolonged*, shows obstruction at the aortic orifice.

A *short, very full, and quickly collapsing pulse like balls of blood* shot under the finger, is met with in regurgitation at the aortic opening. Its fullness is the result of the great accumulation of blood in the enlarged ventricle, and the great force with which it is expelled, while its sudden collapse is the result of the

insufficiency of the semi-lunar valves, which are unable to prevent regurgitation.

The so-called "paradoxical" pulse of Kussmaul, is one which intermits either partially or entirely, with each inspiration. It is not at all dependent upon heart failure, but is supposed to owe its origin to constriction of the ascending portion or arch of the aorta by fibrous bands, the remains of a previous pericarditis, and which are drawn upon by the elevation of the sternum during every inspiration.

*Intermittency* of action of the heart may be divided into *complete* and *incomplete*. When the ventricle fails to contract at all, through one or more auricular contractions, the intermission is said to be *complete*; when it contracts, but so feebly that the pulsation is felt either not at all or but faintly at the wrist, it is called *incomplete* or *false* intermission. Although Hayden, in his recent great work, affirms that he never had but one case of intermittency in which the rhythm was not disturbed, my experience, has been different, the rhythm being perfect in perhaps the majority of cases. The intermissions are in most cases irregular as to the number of pulsations intervening, yet there are some exceptions, as of every third or fourth beat intermitting.

Intermittency is usually a symptom of grave import, and, according to the accompanying symptoms, is indicative of heart failure from fatty degeneration, atrophy, or dilatation, or, in case of hypertrophy, of some great obstruction to the circulation. Under such cir-



cumstances, it is aggravated by any active exercise, although in some cases it may manifest itself only after *violent* physical exertion. Intermission of the heart's action is not, however, always indicative of structural lesion, as it sometimes occurs as a purely nervous phenomenon.

Next to this in point of seriousness as a sign of heart failure, stands *irregularity in rhythm*. By this is meant a halting of the systole during a portion of the period of a pulsation only. It differs from intermittency in that the latter is a halting through the period of one or more complete pulsations. Irregularity in rhythm is of two kinds: that which is the result of a disturbance of the balance between the two opposing nervous forces of the heart, and that which comes from physical incompetency of its muscular walls to do the work required of them, either in consequence of their own debility, or of some obstruction to the circulation, or both combined. A greater or less amount of physical exertion on the part of the patient will tell us with which of these two kinds of irregularity we have to deal. The latter and more serious variety is aggravated by exercise, and the degree of heart failure may be told approximately by the readiness with which exertion induces it. Irregularity and intermittency are frequently both present in the same individual.

Irregularity in volume of pulse is quite distinct from irregularity in rhythm, and will be referred to under the head of mitral regurgitation.



*Palpitation is tumultuous action* of the heart, with or without violence or increased rapidity, and is generally accompanied by an exceedingly unpleasant sensation. It is an increased effort on the part of the organ to perform its functions. It is an evidence of weakness, not of power; it indicates cardiac debility without great structural degeneration. In connection with organic disease it is found most frequently in dilatation of the left ventricle; but when that dilatation is fully compensated for by hypertrophy, the palpitation ceases. Again, when dilatation passes far into structural degeneration, there is no palpitation, probably because the walls are incapable of being excited to violent effort.

But palpitation much more frequently exists *without organic disease*, and in this case the debility is purely of a nervous character. Occurring under such circumstances, it is readily curable by the properly selected Homœopathic remedies, while that which is met with as an accompaniment of dilatation, can at least be greatly relieved by their administration.

An *increased area of dullness* on percussion is often a symptom of great diagnostic value, yet an emphysematous lung may in any case prevent its manifestation. Increased dullness *downwards* and to the *left* is indicative of hypertrophy and dilatation of the left ventricle. Increased breadth of dullness to the *left* alone, with a rounded outline, points to dilatation of the same part. Dullness which is increased *upwards* and *both* to the *right* and the *left*, is found in extensive

pericardial effusion. If the effusion is but slight, it will be difficult to diagnose.

In persons having thin, lean chest walls, the heart sounds are louder and more distinct than usual, while an extensive fatty deposit has an opposite effect. A solidified lung will transmit the sounds farther than will a healthy one. If, the lung being sound, they can still be heard in the back, it should at once suggest ventricular hypertrophy. The valvular element of the first sound is exaggerated when the ventricular walls are thin and dilated, but structurally sound, and especially is this true when the heart is unduly excited.

*Exaggeration of the first sound over the ensiform cartilage* denotes pulmonary obstruction, which may be the result of left side disease or of some pulmonary affection. It is produced by the violent closure of the tricuspid valve by a dilated and hypertrophied right ventricle.

Exaggerated *second sound* is not so frequent at the aortic as at the pulmonary orifice. Exaggeration of the *pulmonary* second sound was considered by Skoda as pathognomonic of mitral obstruction, but Hayden has only seen this symptom in fifteen out of sixty-three cases. In my judgment it is only indicative of some obstruction to the pulmonary circulation, and consequent right ventricular hypertrophy. It is produced by the recoil of the pulmonary artery, and, when that vessel is healthy, corresponds in degree to the force of the contraction of the right ventricle.

There may be *suppression* of one or more of the

sounds of the heart. The *first* sound is suppressed in the softening of typhus fever, and nearly or quite so in the later stages of fatty degeneration, in which cases it is indicative not only of loss of power in the ventricles, but of the inability of the walls, from their change of structure, to transmit the sounds as in health.

The *second* sound is suppressed at both the pulmonary and aortic orifices in Asiatic cholera, while in emphysema of the anterior portion of the lungs, and in cases of extensive serous effusion into the pericardial sac, both the first and second sounds are more or less weakened.

The sounds of the heart may be *doubled*. Doubling of the *first* sound is most likely produced by the muscular element preceding the valvular in point of time, and is not a very common occurrence.

Doubling of the second sound is the result of one set of semilunar valves closing before the other. It is often found in obstruction at the mitral orifice, and sometimes in connection with other lesions. The diagnostic value of this reduplication certainly is not great.

*Murmurs*.—Cardiac murmurs are of two kinds: endocardial, and pericardial. The former, which will be first considered, may be obstructive, regurgitant, or hæmic. They are produced at the four great orifices of the heart, and while these orifices are all included within a space less than two inches in diameter, in either direction, their several murmurs are



radiated outwards, so as to be somewhat widely separated, being heard most distinctly at the points of maximum intensity of the corresponding normal sounds.

Given a knowledge of the anatomy and physiology of the heart, a little reflection as to the course of the normal blood currents will aid very materially in understanding the various murmurs.

A *systolic* murmur heard *loudest* at the *apex* is significant of mitral regurgitation, the mitral valve being, for some reason, insufficient to prevent a portion of the blood from flowing backwards into the left auricle. It may be heard nearly all over the precordial region, and often considerably beyond the apex, and at the inferior angle of the left scapula. It occurs synchronously with the ventricular impulse, and commonly replaces the valvular element of the first sound. It corresponds somewhat in intensity to the energy of the ventricular contraction, and in cases of extreme debility of the heart walls, as in advanced cases of fatty degeneration, it often becomes entirely inaudible.

A *systolic* murmur heard loudest over the ensiform cartilage, is tricuspid regurgitant, and results from insufficiency of the tricuspid valve.

A *harsh systolic* murmur with its maximum intensity at the right hand border of the sternum, in the second intercostal space, is aortic obstructive, the aortic orifice being so constricted as to impede the passage of the blood from the left ventricle into the aorta. This constriction, with the roughness of the

valves which is generally present, produces a murmur of varying pitch and harshness, heard nearly the whole length of the sternum, frequently well down to the apex, and upwards and to the left along the arch of the aorta, and into the carotids, but *always loudest at the point named*.

A *systolic* murmur heard loudest at the left hand border of the sternum, and about the upper border of the third costal cartilage, and limited in extent to this immediate neighborhood, is at the mouth of the pulmonary artery, and may be obstructive, but is generally hæmic.

A *presystolic* murmur at the left apex, is mitral obstructive. It is generally harsh, while the mitral regurgitant is soft and blowing. The obstructive murmur is produced by narrowing of the mitral orifice, and is generally not quite so widely diffused as the regurgitant. It is the diastolic apex murmur of the older writers, but cannot consistently be called such, as it occurs during, and results from, the active contraction of the auricle, just before the ventricular contraction, and extends up to the first sound. It is very frequently found associated with a mitral regurgitant murmur.

A *diastolic* murmur at the base of the heart, is loudest at the aortic orifice, and is aortic regurgitant. The aortic semilunar valves being incompetent—the recoil of that great vessel forces a portion of the blood backwards into the ventricle, and thus is pro,



duced a murmur which is strictly diastolic in time, and replaces the aortic element of the second sound.

For greater convenience in studying these murmurs, the following table is presented:

VALVE.	MURMUR.	TIME.	CHARACTER.	POINT OF MAXIMUM INTENSITY.
Mitral.	Regurgitant.	Systolic.	Blowing.	Apex of left ventricle, and diffused.
Tricuspid.	Ditto.	Ditto.	Ditto.	Ensiform cartilage
Aortic.	Obstructive or hæmic.	Ditto.	Obstructive rasping; hæmic blowing.	Right border of Sternum, in second intercostal space.
Pulmona'y	Ditto, or hæmic.	Ditto.	Obstructive rasping; hæmic blowing.	Third left costal articulation, upper border.
Mitral.	Obstructive.	Presystolic.	rasping.	Apex of left ventricle, not widely diffused.
Aortic.	Regurgitant.	Diastolic.	Blowing.	Aortic orifice as before described.

The various single endocardial murmurs have been classified in the order of their frequency, thus: mitral regurgitant, aortic obstructive, aortic regurgitant, mitral obstructive, tricuspid regurgitant, pulmonary obstructive, pulmonary regurgitant, tricuspid obstructive. The last two are so seldom met with as scarcely to be worthy of mention, and for this reason I have omitted them from the above classification.

Two or more endocardial murmurs are often combined. The following are some of the most frequent combinations, classified in the order of their frequency:

1. Aortic obstructive and aortic regurgitant.
2. Mitral obstructive and mitral regurgitant.
3. Aortic obstructive and mitral regurgitant.
4. Mitral regurgitant and tricuspid regurgitant.

## 5 Aortic regurgitant and mitral regurgitant.

We occasionally meet, besides these, with a post-systolic murmur at the apex, significant of incipient mitral regurgitation, and occurring just after the first sound, during the short pause.

A post-diastolic murmur may be sometimes met with at the right base, significant of incipient aortic regurgitation. It occurs immediately after the second sound.

*Hæmic Murmurs.*—According to Hayden, all endocardial murmurs dependent upon the quality or quantity of the blood, are basic and systolic, and much more frequently heard at the aortic orifices. They are distinguished from organic murmurs at the same orifice, by the quality of the murmur, which is generally blowing, by the accompanying symptoms, and the history of the case. The differential diagnosis will be given more particularly on a future page.

*Dynamic Murmurs.*—Not all cases of mitral regurgitation are the result of disease of the valves. The left ventricle may be irregularly dilated, and the tendinous cords attached to the papillary muscle springing from the dilated portion, may thus draw one of the flaps out of its position at the acme of ventricular systole, so as to prevent it from closing properly, and regurgitation with its attendant murmur is the result.

The apex murmur, so frequently heard in typhus fever is an example of this—

*Venous murmurs or venous Hum,*—These are heard in

the larger veins in certain conditions of the blood as anæmia and hydræmia, are generally continuous, but increased during each pulsation in the accompanying arteries, increased by slight pressure, as with the stethoscope, and totally suppressed by firm pressure, either above or below. They are heard more frequently in the internal jugular than anywhere else, and next, in the external jugular. In most cases, murmurs of either of the three varieties last mentioned are readily amenable to treatment.

*Pericardial Murmurs.*—Liable to be confounded with endo-cardial murmurs, are *pericardial friction sounds*. These are generally to and fro in their character, and heard near the base of the heart. The first element is systolic, being synchronous with the first sound of the heart; the second post-diastolic, occurring immediately after the second sound. If single, it is generally systolic. A pericardial murmur near the apex is quite rare, and when heard is usually soft, single and systolic. These friction sounds are more prolonged than the normal sounds of the heart, and are neither as strictly rhythmical nor as constant, as endocardial murmurs. They are increased in intensity by firm pressure with the stethoscope, which has no influence on sounds produced within the heart, and are liable to variations in intensity, or even to entire suppression for the time, with the changing position of the patient. They are not diffused beyond the point of their production, and are accompanied by tactile fremitus, which sometimes

amounts to a genuine *fremissement cataire*, or purring thrill.

*Pleuritic friction sounds* might lead to confusion, but are generally distinguished from both pericardial and endocardial murmurs by their rhythm, which is that of respiration instead of cardiac action. Holding the breath causes the murmur to cease. Now and then, however, we are likely to meet with a case in which that portion of pleura which is reflected upon the outside of the pericardium becomes inflamed, and here we may have a pleuritic murmur of cardiac rhythm, but even this is generally intensified near the termination of each full inspiration, and heard only at the apex and along the left lateral limit of cardiac dullness. Flint records a case in which such a murmur was diffused over a large portion of the precordium, but the probability of an exo-pericardial pleuritic friction murmur being thus diffused in any given case, is so small as scarcely to be worthy of consideration.



## CHAPTER IV.

### EFFECTS OF AN OBSTRUCTED CIRCULATION.

As, in the vast majority of cases, the manner in which cardiac diseases prove fatal is by the long continued and ever increasing obstruction to the circulation, with the effects which such obstruction has upon the different organs, much repetition may be saved by considering those consequences of obstruction before proceeding to the study of the various cardiac diseases individually. This short chapter, then, will treat not particularly of the consequences to the heart itself, but of such as are felt by the other organs of the body.

In affections of the aortic valves, the chief danger, as long as the mitral valve remains perfect, is from rupture of the cerebral arteries, forming genuine apoplexy, as the blood regurgitating into the left ventricle, added to that coming in from the auricle, so increases the amount of that fluid, that when the hypertrophied ventricle contracts, it produced too great a strain upon the arterial system. In aortic obstruction, there is always some danger of sudden death from paralysis of the left ventricle, during and after violent exertion. In aortic insufficiency, even while the mitral valve remains intact, there may be more or less engorgement of the lungs, as the regurgitation partially fills



the ventricle, and must to some extent prevent the proper emptying of the auricle and pulmonary veins. When, however, the mitral valve once gives way, either primarily or as a consequence of aortic disease, we must, in proportion to the amount of regurgitation into the auricle, sooner or later have the evil consequences of this backward working, or as German pathologists call it, "ruckwirkung." The auricle being already filled by the regurgitation, the onward flow of blood from the pulmonary vessels is prevented, and they become dilated; the stagnant blood in these vessels impairs the function of the right ventricle, and hypertrophy and dilatation are set up there also. Next, the tricuspid valve gives way, the right auricle is in its turn dilated, and the entire venous system becomes engorged. The result of this venous and pulmonary engorgement, is congestion of the various organs of the body, with impairment of function and increase of their connective tissue, followed by atrophy and degeneration.

As left side failure precedes right side failure, so the lungs are the first to suffer from the consequent stagnation, and we have dyspnoea which soon becomes worse in paroxysms, worse on lying down and after every exertion, frequent attacks of bronchitis, and when the pressure is very great, as between left side valvular disease and hypertrophy of the right ventricle, hæmoptysis may result, or even pulmonary apoplexy. Emphysema is often seen, the dyspnoea being so great as to cause rupture of the air cells, while, in

the last stages, pleuritic effusion is not uncommon. One of the most constant symptoms is cough, which is at first dry, and afterwards attended by a profuse frothy, serous, or very thin mucus expectoration, sometimes of sweetish taste and tinged with blood.

With, and to a limited extent before the giving way of the tricuspid valve, the venæ cavæ and their branches become distended with stagnant blood. This distention in turn renders the venous valves, which normally prevent the backward flow of blood, incompetent, and every systole of the right ventricle produces a regurgitant wave which is felt in the larger veins, and, German pathologists say, in the liver also. There certainly is in tricuspid regurgitation a pulsation in the liver, which can be felt through the abdominal parieties, but whether or not this is ever felt without tricuspid regurgitation, or whether it is at all the result of the regurgitation, or is simply the motion of the enlarged right ventricle communicated to the liver through the thin diaphragm, which is almost the only thing which separates the two organs, it is not yet definitely known. However this may be, even before any of the venous valves give way, the symptoms of venous congestion begin to be felt.

Among the first organs to suffer is the brain, which of course becomes more affected as the disease progresses. We have here, as in other organs at this and later stages of the disease, venous congestion with deficiency of arterial blood, with consequent increase of parenchyma, followed by atrophy, with effusion into

the meninges; and as symptoms of the pathological condition, dull headache, dizziness, feeling of fullness in the head, confusion and weariness of mind, irritability, instability, and sleep disturbed by anxious, horrible dreams. The face is apt to be dark and congested in appearance, and the lips blue.

Not the least important among the results of this venous engorgement, are the affections of the digestive organs, for here we have all the symptoms of gastric catarrh, with indigestion, acidity, violent and excessive eructations, heartburn, nausea, vomiting of food, and a constant feeling of fullness even when the stomach is empty. We have also constipation or diarrhœa, or the two in alternation; and, as a very common attendant, the mucous membrane of the rectum becomes so congested as to produce profusely bleeding piles.

The liver and spleen also participate, the latter organ being felt, enlarged and projecting from under the ribs, while, especially in the liver, the connective tissue is increased, followed in time by atrophy; and as symptoms of this condition, jaundice, with deficiency of bile in the intestinal canal, and highly colored urine.

The kidneys undergo a similar change of structure to that which takes place in the other organs, and with equal impairment of function, the urine being scanty, highly colored, and containing more or less albumen and tube casts. The diminution in quantity of the urine is not, however wholly dependent on the



change of structure, but, in a great degree, is the result of diminished arterial tension, there being a scarcity of blood on the arterial side, so that anything which strengthens the heart's action and renders it more efficient, increases for the time the amount of urine.

The mucous membrane of the bladder becomes congested, and so much so as to produce all the symptoms of a genuine vesical catarrh, with burning and pain in the organ, and frequent emission of scanty urine, with violent strangury.

The female generative organs usually suffer more or less, the patient being troubled with leucorrhœa of thin watery mucus, profuse menstruation, and, in many cases, metrorrhagia, with a disposition to recurrence of the sanguineous discharges after the climacteric has been passed. In men are found congestion of the prostate, nocturnal emissions, and in the last stages, probably hydrocele as a result of this congestion.

As time goes on, the system at large becomes more or less seriously affected. The circulation being feeble, we have coldness of the exterior, and of the extremities in particular. The cellular tissue in different parts becomes œdematous, and when that condition becomes general, the patient is said to have anasarca. When this is seen under the eyes, it is considered as an indication that the kidneys have become implicated, and the more these organs are involved, the greater is the tendency of the œdema to become general. By appropriate means, this dropsy can easily be removed in its

earlier stages, but it is certain, sooner or later, to return, and each time it returns it is a little worse and a little more obstinate than before, until finally, in the last stages, when the serous membranes have become implicated in the effusion, nothing seems to be of much avail. The appearance of dropsy is often at first a relief to the patient, the dyspnœa and all the other consequences of congestion being thereby greatly diminished; or a hæmorrhage from the lungs or bowels or other organ, or an attack of profuse watery diarrhœa, will be attended with the same temporary benefit.

In the last days, when the patient's kidneys no longer act with any efficiency, when the heart is scarcely able longer to beat, and respiration has become so difficult as to be but very imperfectly performed, the blood is not properly purified, and carbonic acid poisoning supervenes, with its attendant drowsiness. This drowsiness is often terrible; for, as the patient falls into a slumber, the respiration gradually fades away until it ceases entirely, and it would seem that death must come, but suddenly and with alarm, he awakes to make another effort to breathe, again to fall into the same troubled sleep. But this terrible suffering cannot last long, for death, often prayed for, soon comes to his relief.

Another very common accompaniment of these later stages is the symptom of Cheyne, the patient breathing in undulations of twelve or fifteen respirations,



now fading away into a complete intermission, now gradually increasing into a full inspiration.

I have mentioned many symptoms of obstructed circulation, but it must not be supposed that every or even any patient has all these symptoms; nevertheless, the general character of the manifestations is the same in nearly all cases, from whatever cardiac lesion the obstruction may result, one case being lighter, another more serious, one making slower progress, so that a patient may live for a great many years with but little inconvenience, while another is more acute and passes rapidly on to a fatal termination. Yet, however severe the cardiac disease, it is not always *that* which kills, but it increases the mortality of other diseases. As an example of this, a moderate attack of pneumonia, which under other circumstances would not be called a particularly dangerous disease, when occurring in an advanced stage of heart disease, when the pulmonary engorgement is very great, is almost certain to result in death; and inflammations of other organs are correspondingly difficult of treatment; besides, as the general health is impaired, new diseases are more readily set up than under other conditions.

## CHAPTER V.

### PERICARDITIS.

This disease may be divided into the acute and the chronic form. Acute pericarditis is usually the result of acute rheumatism, being present in probably one-sixth of the cases of that affection, and generally manifesting itself before the tenth day; very seldom later than fourteen days. Sometimes, indeed, it may show itself as the beginning of the disease, or the attack of rheumatism may in rare cases spend its whole force upon the pericardium, as it sometimes does upon other single parts. Acute pericarditis occurs occasionally, also, as a complication of other affections besides rheumatism, as pneumonia, pleurisy or chronic Bright's disease, or in the desquamative stage of scarlatina, or it may result from wounds, from abscesses in neighboring organs, or from the suppression of an exanthem, or idiopathically, from a too sudden change of temperature, or even from tuberculosis or cancer. The rheumatic form is generally found in acute rheumatism, seldom in chronic and as acute rheumatism is a disease of early life, pericarditis is also most frequently found in the young. It is more frequently met with in the fibrous than in the synovial form of rheumatism. That which is the result of dyscrasic affections, or of chronic

kidney disease, belongs rather to those who have passed the meridian of life. It perhaps occurs in six per cent of the cases of chronic Bright's disease.

Again, inflammation of the pericardium may be divided into the diffuse and the circumscribed, the number of cases of the diffuse very largely preponderating. When circumscribed, it is most frequently found about the base of the heart and around the origin of the great vessels, although it may occur at other points.

Three stages are usually recognized. First, the stage of dryness, or suppression of the normal secretion of serum. Second, the stage of effusion of fibrine or lymph. Third, that of serous effusion.

It is seldom recognized during the first stage, generally being at that time entirely devoid of distinctive physical signs, and the subjective symptoms being decidedly equivocal. We usually have at that time, more or less pain in the cardiac region, with sharp and accelerated impulse, increased activity of the heart, and the sounds clear and ringing. Careful auscultation also discloses a slight rustling noise proceeding from the dry pericardium, different from the friction sound of the later stages. There is now no tactile fremitus, for the two layers of serous membrane are yet gliding smoothly upon each other. The circulation will generally be found at from 100 to 120, with proportionately quickened respiration, and a dry, harsh, ringing cough, which can only be ac-



counted for by reflex irritation. The duration of this stage is from 12 to 48 hours.

Post mortem examination shows the membrane dry and red from injection of the vessels beneath.

In as much as the second and third stages are very intimately connected, I shall not attempt to draw a sharp line between them, but shall consider them together as one, the second stage. This comprises the period of the effusion of a substance of greater or less quantity, from the most minute to a quart or more in extreme cases, and possessing various degrees of density, now consisting of almost pure lymph of great elasticity, and so dense as to glue the two layers of the pericardium together, so that when separated they look like tripe, and again of almost pure serum; again it may be composed of these two substances in more nearly equal proportions, the one or the other generally predominating, or it may be hæmorrhagic in character, as in scurvy, while in a few cases it is purulent. It varies also in color: the fibrinous being generally yellow or reddish yellow, while the serous may be a bright yellow or transparent, greenish, or reddish brown. The hæmorrhagic is of course the color of blood, and is most frequently met with in scurvy, although, according to Bauer this variety of pericarditis may occur in otherwise perfectly healthy subjects.

The fibrinous portion of the effusion is generally thrown out first, and afterwards, if at all, the serous, which, after remaining an indefinite length of time,



varying from a few days to several weeks, is afterwards reabsorbed, leaving the fibrinous portion.

During the stage of effusion, which constitutes the principal duration of the disease, is the only time at which a diagnosis can be made with absolute certainty, and even here the best of diagnosticians have sometimes erred after repeated examinations, and only discovered the mistake at the autopsy. The only signs to be absolutely relied on, are those elicited by a physical examination, yet the subjective symptoms are not without value. They are all, however, more or less equivocal, because they may be each produced by some other disease than pericarditis, as for instance, some affection of the lungs or pleura. They are the more difficult to describe, from the fact that it seldom exists alone, but usually in connection with acute rheumatism. One of the most constant symptoms is pain in the precordial region, not usually very severe, but aggravated by firm pressure, and by taking a deep breath. If the lung or pleura be implicated in the inflammation, the pain is of course more severe. The patient usually has a peculiar look of suffering and anxiety for relief, which appearance would naturally be looked for in inflammation of the joints as well, but in pericarditis is much more marked. In the more severe cases, when the heart shows signs of failure, either from the extension of the inflammation to its own substance, or from pressure of the effused fluid, or from whatever cause, we may find aggravated dyspnoea, severe oppression at the precordium, loss of pulse, anguish and fear of

death. We have also blue lips, irregularity and palpitation of the heart, headache, and sometimes nightly delirium; but let it be remembered that these last mentioned signs are not *peculiar* to pericarditis, being only symptomatic of the cardiac debility which accompanies it, and found in other conditions as well.

*Physical signs.*—Inspection yields no important results, except in cases of extensive serous effusion in quite young subjects with yielding chest walls, in which the precordium is sometimes seen to bulge forwards very decidedly. The absence of energetic action serves to distinguish between this and hypertrophy. Bulging of the intercostal spaces is not seen unless the effusion be very great. The absence of visible pulsation at the apex in a thin subject is indicative of either serous effusion or cardiac debility.

A very common symptom is tactile fremitus, which seldom amounts to a genuine purring thrill. This is felt on applying the palm of the hand to the cardiac region, and results from the vibration caused by the gliding of the two roughened and sticky surfaces upon each other. It is to the touch, what the friction sound is to the ear, both being produced at the same time and in the same way, but it is not so constant a symptom as the other. From the manner of its production, it is evident that it cannot be felt when by any means the two layers of pericardium are prevented from coming in contact with each other; thus, the effusion of a considerable quantity of serum causes it to cease, while, with the absorption of the serous effusion, it re-

turns. But fremitus, although a valuable symptom, is not pathognomonic of pericarditis, as it has been met with as an accompaniment of all organic endocardial murmurs except the basic regurgitant (aortic and pulmonary), most frequently, however, with the mitral obstructive, in which case it is pre-systolic in time. Fremitus of endocardial origin, however, is more apt to be exaggerated into a purring thrill. Again, by palpation we discover the position of the apex when the effusion is not too great, and thus we are assisted in ascertaining whether or not dilatation be present. When the effusion is great, the apex cannot be felt while the patient is lying upon the back, nor in the majority of cases yet even when standing erect, but if he leans very decidedly forward, or lies upon the face or left side, the heart, being heavier than the fluid which surrounds it, comes in apposition with the chest wall, and its pulsation is again palpable, although not so distinctly as where there is no effusion. Turning on the left side of course carries the apex to the left, yet it has an advantage over the prone position, in that it better admits of physical examination.

The lost impulse, in order to be *pathognomonic* of pericardial effusion, must be *more* than barely perceptible when thus restored by change of position, for an apex beat which is lost in consequence of fatty degeneration, may be again feebly perceptible when the patient turns upon the face.

*Percussion* yields only negative results as long as there is but slight serous effusion, after which, there is



an increased area of deep dullness, the area of superficial dullness not generally varying in direct proportion to the amount of the effusion. Sometimes when the pericardial effusion is very great, the lungs may be so displaced as to greatly increase the area of superficial dullness, while at other times, the effusion being equally great, it is not increased in area at all, as the lungs have not been displaced, having been previously bound down by pleuritic adhesions.

The effusion naturally seeks the lowest level, so that deep dullness assumes, to a limited extent, the form of a triangle, the base of which is below and the apex at the left base of the heart, and sometimes extending upwards as far as the clavicle. If the quantity of serum is not so great as to prevent the impulse from being felt, the dullness will be found to extend to the left, beyond the apex. This is a sure sign of pericardial effusion, and is most readily discernible when the patient is sitting upright. If the quantity of the fluid is great, the dullness may extend to the right, beyond the sternum. It seldom extends downwards, however, beyond the sixth rib, as the diaphragm is not apt to be displaced. The base of the triangle can be determined by ascertaining the lower limit of dullness at the extreme left, and drawing a line from this point transversely to the right hand border of the sternum. The fact of hepatic dullness being continuous with the cardiac, renders it impossible to ascertain the lower limit in any other way. "When the patient lies down this triangle assumes more of a rounded form." (May-



den). Turning upon either side of course increases the dullness in that direction. Care should be taken that a lung solidified anteriorly should not be mistaken for a pericardial effusion.

If percussion fails to distinguish between enlargement of the heart and effusion into the pericardium, inspection, palpation and auscultation come to our aid, for in the former the heart's action, instead of being concealed, is more than naturally manifest.

*Auscultation.*—The second stage is generally ushered in by the appearance of a pseudo-murmur, or genuine friction sound, caused by the rubbing of the two layers of pericardium upon each other. It is usually harsh in quality and situated about the base of the heart, although it may be at other portions, or even all over that organ, and is increased by firm pressure of the stethoscope. There is, besides, a sort of unsteadiness about it, an irregularity of site, rhythm and intensity, and when produced upon the anterior or lateral surfaces of the heart, it has a sound of superficiality not possessed by an endocardial murmur. It is most frequently double, systolic and post-diastolic, but may be single and systolic, or triple, a faint pre-systolic sound being added to the other two. Change of position, as lying down after sitting, or turning to one side or the other, is likely to change the rhythm, or to increase, diminish or entirely suppress one or all of its elements. It is not widely diffused, not being heard beyond the pericardium, and generally over only a small portion of it, and is liable to changes of site in

short periods of time, being heard one day at one portion of the precordium, and the next day at another portion.

It is the most constant and trustworthy sign of pericarditis, there being but few cases in which it cannot be heard at least a portion of the time, those cases in which it is altogether absent generally being characterized by the effusion of a large amount of serum and very little fibrine. The friction sound once established, the effusion of a sufficient quantity of serum to separate the pericardial surfaces causes it to cease, commonly to reappear with the absorption of the fluid. It usually continues thereafter until adhesion takes place by the organization of the effused lymph. Extreme cardiac debility sometimes renders it inaudible.

To distinguish between pericardial and pleuritic friction sounds, let the patient hold the breath. If the sound ceases it is pleuritic. Now and then, however, a pleuritic murmur may be cardiac in rhythm. In this case, it proceeds from that portion of the pleura which is reflected upon the pericardium, and may still be distinguished by being situated at the left apex, or at some point along the left lateral limit of cardiac dullness, and not diffused over the precordium.

But that with which a pericardial friction sound is most likely to be confounded, is an endocardial murmur. The following table of comparison will help to distinguish between the two:

## ENDOCARDIAL MURMUR.

Constant in rhythm.

Constant in situation.

Change of position has no effect.

Constant in character.

Pressure does not affect it.

Respiration has no effect.

More frequently single.

When double, if both murmurs are basic, systolic and diastolic. If at the apex, pre-systolic and systolic.

Deeply seated.

Murmur can still be heard when ear is removed a little distance from the stethoscope.

Generally more widely diffused.

## PERICARDIAL FRICTION SOUND.

Irregular in rhythm.

Liable to change of site in short periods of time.

Change of position increases, diminishes or suppresses it, or changes its rhythm.

Irregular in character.

Pressure increases it.

Often increased or diminished with each respiration.

More frequently double, sometimes triple.

Generally near the base, but may be at the apex or other points. When double, is systolic and post-diastolic.

Generally seems near the surface.

Generally renders friction sound inaudible.

Not usually so widely diffused.

The normal first sound of the heart during serous effusion is not so distinctly heard as it would otherwise be, and if the amount of fluids is great, and the heart walls at the same time considerably debilitated, as by extension of the inflammation to the muscular substance, may be completely inaudible. If, however, the heart muscle is not debilitated, and the heart sounds are obscured by the effusions, the pulse will be stronger than the heart sounds would seem to warrant.



Implication of the heart walls in the inflammation, is likely to render the pulse feeble and irregular or intermittent.

Again, the pressure of a large quantity of fluid within the pericardium may prevent the auricles, with their thin and yielding walls from expanding sufficiently during their diastole, so that they are not properly filled, and hence there is a deficiency in the quantity of blood thrown into the arteries, and we accordingly have a pulse which is small and feeble, although not necessarily irregular or intermittent. Bauer (Ziems-sen, Vol. VI), thinks the pressure of the effused fluid upon the beginning of the great vessels likely to prevent their proper filling during systole, thus producing a small and feeble pulse, but this is not at all probable, as the pressure is equal at all points, that upon the ventricles neutralizing that upon the great vessels.

The imperfect filling of the auricles in extreme degrees of effusion, may, according to the same author, produce congestion of the pulmonary and venous systems, with cyanosis and even general dropsy, but the great cause of the venous and pulmonary stagnation, is debility of the heart walls.

Now and then, a case is met with in which this venous congestion gives rise to severe cerebral symptoms. Even wild delirium may be present, and the cerebral symptoms may so far predominate over the cardiac as to lead to the supposition that the patient is suffering from an attack of meningitis. In such cases, very



close observation on the part of the physician is necessary, in order to know to what point to direct his attention, and not to be treating a pericarditis for a meningitis, which would require a widely different treatment, and have a very different prognosis.

*Terminations.*—The duration of acute pericarditis, when left to itself, is usually from one to two weeks, at the end of which time, if death does not take place, recovery begins, or it may, in some cases, pass on into the chronic form. From its nature it may have many different terminations. The inflammation may never proceed beyond the stage of injection and dryness, and thus complete restoration may take place, or if there is an effusion consisting of almost pure serum, it may be completely absorbed, and a perfect cure be the result. The fibrinous portion of the effusion may be either partially or entirely absorbed, but not so readily nor so rapidly as the serous; but more frequently, adhesion takes place by organization of the effused lymph. Even a sanguineous effusion may be entirely absorbed, but more frequently it terminates fatally; not, however, from the character of the effusion, but as a result of the accompanying conditions.

The effused fluid may gradually take on a purulent nature, and abscess result, which will be likely to break into the pleural sac, (although this is a rare termination) or the liquid portion may be absorbed, leaving the solid constituents, which gradually assume the shape of cheesy or calcareous masses. The heart muscle may become infiltrated with the serum of the

effusion so as to weaken it and produce dilatation, which may afterwards be compensated for by hypertrophy, or the pressure of the effusion may produce atrophy.

The inflammation often extends into the heart muscle itself, and the part so inflamed is likely afterwards to take on fatty degeneration.

The most frequent termination of pericarditis is in adhesion of the pericardium, mentioned before, and this may be partial, in one or more portions of the heart's surface, the adherent portions elongating into bands as is so frequently the case in pleuritic adhesions, or in streaks, dividing the pericardial cavity into different compartments, or it may take place over the entire surface, completely obliterating the sac. It may also extend to the pleura, the pericardium and pleura being united, and drawing the lung down over the heart and diminishing the area of normal superficial dullness. Adhesion may follow the effusion of lymph without serous effusion ever having taken place. In such cases, the whole process may be completed in twenty-four hours from the time adhesion begins. As it progresses the friction sound gradually diminishes in intensity, and becomes more irregular in rhythm, and single and systolic, and its area diminished, until finally it ceases altogether with the completion of the process of adhesion. The adhering mass occasionally undergoes calcareous degeneration, thus forming one or more hard bony plates upon the surface of the heart, or that organ may sometimes be almost completely enveloped in the

bony covering, so that it would seem impossible for it to perform its functions.

To diagnose a pericardial adhesion with absolute certainty, with the present state of our knowledge, is impossible, yet, if there is a positive knowledge that the patient has had pericarditis, the apex is permanently displaced upwards, and the first sound is resolved into its two elements, with the impulse diminished in force, it may be presumed to be present. (Hayden.) Its existence can only be established with certainty, when a systolic retraction of the lower portion of the sternum along with the left inferior costal cartilages, and a diastolic recoil of these parts, takes place. (Rosénstein.) Absence of apex beat is only suggestive of it. If the area of superficial dullness is not diminished by a full inspiration, and at the same time the intercostal space at the point of apex pulsation is drawn inwards at every systole, it is pretty good evidence that the adhesion has taken place, not only between the two layers of pericardium, but also between that membrane and the pleura.

It has been supposed that pericardial adhesions are prolific causes of hypertrophy, but this is evidently an error, for in nearly all cases in which the two occur together, there is something else connected with it, amply sufficient to explain the hypertrophy. Hayden quotes Wilkes as saying that simple pericardial adhesions produce no visible ill consequences, but this is altogether too sweeping. Among other evil effects is interference with the heart's action. If this interference



is but slight, and the coronary circulation is not interfered with, the result may be a slight hypertrophy; but even then dilatation is more common. If the coronary arteries are constricted by the adherent pericardium, the circulation within the heart walls is obstructed, and atrophy and degeneration are likely to result. Thus, it will be seen that in many cases of pericardial adhesion, we are liable to have cyanosis, dropsy, and all the other consequences of an obstructed circulation.

When pericarditis occurs a second time, it is usually not so violent as a first attack, and in consequence of the adhesions that have previously taken place, the friction sounds do not so clearly indicate the limits of the inflammation, while their rhythm is greatly interfered with.

The chronic form is not so easy to diagnose as the acute, and, according to Hayden, in constitutions young and vigorous, is certain to result in hypertrophy, while in those of a more feeble habit, dilation results. The same author says of the so-called "milk spots," that he considers them to be due to circumscribed and aborted pericarditis, of a sub-acute character. I see nothing irrational in this explanation, yet there are no means of demonstrating it to a certainty. They are most frequently found upon the anterior surface of the right ventricle.

Perhaps the most frequent complication of pericarditis is endocardial inflammation, the latter being present in perhaps about one-half of the cases, not



however, as a consequence of the pericarditis, but dependent upon the same cause. This, of course, modifies the symptoms, both subjective and objective. The pulse is usually more rapid, and there is commonly, in addition to the friction sound, a murmur at the apex, from mitral regurgitation. This murmur is systolic, and soft and blowing, in contradistinction to the harsh friction sound of pericarditis. In a little time also the left ventricle becomes dilated and perhaps hypertrophied; but it must not be supposed that every mitral regurgitant murmur heard in pericarditis or elsewhere, is an indication of endocardial disease, for frequently the valve is prevented from closing by the giving away of a portion of the ventricular wall during systole, and regurgitation, with its attendant murmur, is thus temporarily produced.

*Prognosis.*—The prognosis is not always easy, and depends not only upon the character of the effusion, whether serous, fibrinous, hæmorrhagic or purulent, (in the last two of which the prospect of recovery is small,) but on the patient's constitution, and the nature of the affection which it accompanies or succeeds. In simple pericarditis, that which results from Bright's disease is generally acknowledged to be the most fatal, while that which accompanies acute rheumatism is attended with but little danger. That resulting from wounds of the pericardium is not necessarily always fatal. Endocardial complications render the disease much more serious, especially in its after effects.

If the heart muscle is involved, the part so inflamed

is likely to take on fatty degeneration, while if this myo-cardial complication is extensive, there is great danger of speedy death during the progress of the active inflammation. Simple, uncomplicated, acute pericarditis, is not generally attended with much danger to life.

*Treatment.*—From the yet exceedingly limited amount of knowledge we possess of the action of medicines upon the heart and its membranes, and the almost total absence of physical signs in their pathogenesis, it is obvious that any chapter on the therapeutics of cardiac diseases must be very imperfect. Although our literature possesses many clinical reports of this class of affections, but little reliance is to be placed in most of them, from the fact that the average physician of whatever school, knows perhaps less of the diagnosis of diseases of the heart than of any other organ; still, enough is known to demonstrate the superiority of Homœopathy over all other schools, in cardiac affections, and this superiority depends not simply upon the general harmlessness of Homœopathic medication, which is not by any means unimportant when compared with the injury often done by injudicious treatment, but more especially upon the fact that in the main, our treatment is direct and specific.

The object of treatment of pericarditis is, first, to subdue the inflammation. Second, to remove its effects, as to promote the absorption of the effused fluid, to strengthen the heart walls and to improve the general health. If we do not succeed in cutting short

the attack at once in the first stages, we may at least render it shorter and much less severe and dangerous than it would otherwise be. It is very important to recognize its existence at as early a day as possible, and right here let me say that no case of acute rheumatism should be treated without examining the heart at each visit; but while doing so, care should be taken that the breast be not too long exposed to the cold, lest an inflammation be set up where none existed before.

The following are the remedies most likely to be found useful. Believing that a few symptoms and those eminently characteristic will be found more useful in choosing the proper medicine, than a host, many of which are unimportant, I shall endeavor to give the indications for each as briefly as possible.

*Aconite* is the remedy most frequently indicated in the first stage and beginning of the second, and will have its best effect if administered as low as the first decimal dilution, a few drops in one half a glass of water, a teaspoonful of which should be given every hour until decided improvement sets in, or until some other remedy is called for. Its use in these doses is limited to the first forty-eight hours or less from the invasion, and here it constitutes the great remedy, far above all others in value, being often capable of cutting short the disease at once.

The indications are: a hard pulse, dry hot skin, and thirst. The pain, if any exist, is cutting, shooting, burning or stitching. The urine is scanty and highly



colored, often burning, the countenance injected, with anxious expression and fear of death. Aconite is indicated in those cases which result from rheumatism, and in the traumatic and idiopathic forms. Those occurring as a consequence of Bright's disease, pyæmia, etc., require something else. It is still sometimes indicated in later stages, with cold skin, feeble, intermitting, and irregular pulse. In this stage it must be given higher, or much mischief will be done.

*Bryonia* comes in at the point where Aconite low, ceases to be of use; that is, during the period of exudation of plastic lymph. Hence almost necessary indications are tactile fremitus and friction sound. Even if there be somewhat increased dullness on percussion from slight effusion of serum, this does not contraindicate *Bryonia*, provided the friction can still be felt and heard, but if the effusion is profuse and the enlargement consequently great, some other remedy must be chosen. Red cheeks, dread of motion, the patient wants to lie very quietly, as every motion causes pain; thirst for large quantities of water; pressive pain in the precordial region; the heart beats violently and rapidly, pulse full, hard and rapid; anxiety; apprehensive of the future; *ill-humored*. These are some of the indications.

*Cactus* may be found useful in the more plastic form, both acute and chronic. Where considerable serous effusion is present, it is not likely to be of use. Among its symptoms are: Periodical attacks of suffocation, with fainting, cold perspiration on the face,



and loss of pulse; palpitation of the heart continues day and night, worse when walking and at night while lying on the left side; dull heavy pain in the region of the heart, increased by external pressure; sensation of constriction in the heart, as if an iron band prevented its normal movement; very acute pain, and such painful stitches in the heart as to cause him to weep and cry out loudly, and with obstruction of the respiration; great sadness, melancholy, irresistible inclination to weep. I have used Cactus in both the 1st and 30th attenuations, and see no reason to complain of either.

*Spigelia* is sometimes indicated in the plastic form, occurring as a consequence of sub-acute or chronic rheumatism, and may then be used either in the early stages or after it becomes chronic. It is also occasionally beneficial in removing the consequences of a previous attack of pericarditis, no inflammation being present.

*Symptoms.*—Dull stitches in the region where the beats of the heart are felt; uneasy strong beating of the heart; the beating can be seen externally through the clothes; palpitation of the heart and anxious oppression of the chest; palpitation increased by sitting down and by bending the chest forward; *vertigo to falling*. Sadness with anxiety.

With the exception of Aconite, all the remedies so far given, being especially useful in the plastic form, are indicated, as a class, by friction, sound and tactile fremitus, and contra-indicated by greatly increased

breadth of dullness when combined with perfect concealment of impulse at the apex.

*Arsenicum*, if indicated, will be likely to find its place in pericarditis resulting from pyæmia or from chronic Bright's disease; or if in the rheumatic form, only in case of extensive serous effusion; therefore with extent of dullness considerably increased both to right and left, and loss of impulse except when leaning forward or lying upon the face.

Among its leading symptoms would be tightness in the precordial region, dyspnœa and palpitation after the least motion, feeble and irregular pulse, skin and extremities pale and cold, great debility. Violent thirst for very small quantities of water, frequently repeated, mouth, tongue and throat dry and burning, I would not advise its use below the 6th attenuation, and should prefer the 30th.

*Digitalis* is particularly indicated in the stage of effusion, very rarely in the first stage, and then only in the medium dilutions. The effusion is not one of plastic lymph, but of serum, and the more decided is this character, the better is the indication for *Digitalis*. There is, therefore, increased extent of dullness, with concealment of impulse, while a friction murmur with fremitus is a contra-indication. When the patient is lying down upon the back, the heart sounds are almost imperceptible, and especially is this true of the first sound. The heart is not only weakened but its sound concealed by the fluid.

If lying down, the patient generally has the shoulders

and head raised considerably to relieve the dyspnœa, which is very troublesome. The pulse is feeble and perhaps irregular and intermittent, and the urine scanty and of somewhat higher color than normal. There is very commonly a sense of faintness or gone-ness at the stomach, and dyspnœa is almost constantly present. The lips are often blue from imperfect oxygenation of the blood.

It may be of use in cases of plastic effusion if the signs of heart failure are very prominent. It should be given as low as the 1st decimal dilution, of which from two to six drops might be given at a dose every hour or two. By this means many a case of pericarditis has been completely and permanently cured. It is also of very decided benefit in renewing the consequences of a previous inflammation in which the heart muscle itself had been involved, and symptoms of dilatation are manifested.

*Sulphur* should not be forgotten, and is valuable in the second and third stages in cases resulting from the retrocession of an eruption, or even in the rheumatic form where the improvement continues for a time and then ceases, in which case a few doses of Sulphur may be found the thing needful. Chronic cases are pretty apt to require it. The chief benefit to be derived from it, is to prepare the way for the action of some other medicine having a more specific affinity for the part affected. A few symptoms are: Palpitation of the heart when going up stairs or when climbing a hill, or without any apparent cause; sensation as if the



heart were enlarged; vertigo when walking in the open air; vertigo in the evening when standing, with rush of blood to the heart. Despondency; great disposition to weep; out of humor, frequent spasmodic jerking in the whole body; burning of the soles of the feet; the patient puts them out of bed. Burning heat on the top of the head.

The above constitute the leading remedies for pericarditis, yet in an occasional case one of the following may be required:

*Arnica*.—Pain in the region of the heart as if it were squeezed together, or as if it got a shock; stitches in the cardiac region; stitches in the heart from the left side to the right. Pulse feeble, hurried and irregular; pleuritic complication; particularly indicated if the attack is the result of a mechanical injury. Use both locally and internally.

*Iodium*.—Complication with croupous pneumonia; purring feeling in region of heart; (Lilienthal) great precordial anxiety, obliging him to constantly change his position; heavy pressive pain constantly in region of heart; palpitation increased by slightest movement.

*Kali carb*.—Swelling like a bag between the upper lid and eyebrows; pinching pains, as if the heart were hanging by bands; heart's action irregular or intermittent; worse at 3 A. M.

*Lachesis*.—Desperate fits of suffocation, she must sit up in bed; feeling of constriction about the heart; cramp-like pain in the precordial region, causing pal-



pitiation, with anxiety; restless and trembling; hasty talking; worse after sleeping.

*Pulsatilla*.—Catching pain in the region of the heart, subdued for the time by pressure with the hand; the patient weeps easily, is thirstless, often changes position, has a loose rattling cough, worse on first going to bed; rheumatic pains, which quickly change locality; suppressed menstruation.

*Rumex cr.*—During rheumatism; burning, stinging pains in the left side of the chest near the heart, when taking a deep inspiration, and when lying down in bed at night.

*Tartar emetic.*—Oppression at the heart; violent palpitation, pulse rapid, weak and trembling, or small and contracted; in complication with pleuro-pneumonia.

*Veratrum vir.*—Faintness and blindness from sudden motions, or when rising from lying, or when walking; better when lying quietly; pulse slow, soft and weak, or irregular and intermittent

*Aspiration.*—In cases in which the effusion is so great as to place the patients life in immediate danger from suffocation or cardiac paralysis, it may be advisable to remove the fluid by means of the pneumatic aspirator, which has the advantage of effecting the removal gradually, while preventing the admission of air into the pleura or pericardium. If the effusion be purulent, the operation becomes so much the more necessary. When properly performed, it is not nearly so dangerous an operation as might be supposed, but if the fluid

is extracted too suddenly, we may hasten the very end we wish to avoid. Just how great the tendency to a refilling of the sac, is not yet known. The puncture should be made in the fourth or fifth inter-costal space, close to the left hand border of the sternum. (Bauer.) Before beginning the operation it is necessary to be absolutely certain that the increased dullness and other symptoms are due to effusion and not to cardiac enlargement. If the symptoms are due rather to cardiac debility than to the great quantity of the effusion, of course aspiration is not usually indicated.

*Hygiene.*—Everything which excites the heart to action will be likely to increase the inflammation; hence, the more quiet the patient is, the better, while in severe cases with feeble and perhaps irregular pulse, it is absolutely necessary that he maintain the recumbent posture continually, not rising for any purpose whatever, nor even turning himself alone in bed, lest the heart be overpowered and paralyzed in the effort to carry on the circulation, and death immediately ensue. The exception to this rule is where the dyspnœa from the effusion or from pulmonary œdema renders lying down impossible.

Now and then a loose pericardial adhesion may be broken up, and the offending fibrinous band absorbed but it is very evident that as a rule pericardial adhesions are incurable. Should however, any troublesome symptoms arise, they must be removed by the appropriate Homœopathic remedies. (See therapeutics of dilatation.)

*Hydropericardium*.—Dropsy of the pericardium is a very different thing from the effusion of pericarditis, being entirely unconnected with any inflammation of the part. The effused fluid may be considerable, or small in quantity. There may generally be anywhere from an ounce to a pound and a half, although in extreme cases much more has been found. About 96 per cent. of its weight is water and the rest solid matter. It not only interferes mechanically with the action of the heart, embarrassing its movements, but the heart muscle frequently becomes infiltrated and softened in consequence.

This affection is occasionally seen in the last stages of Bright's disease, as a consequence of the diffused œdema which is so generally present.

We frequently see it also in the last stages of chronic heart disease, arising in this case from the general venous congestion, in common with the effusions which take place in other serous cavities. It generally increases the patients suffering, and is a sign of the worst import. It is frequently seen also in the later stages of general dropsy.

Perhaps the disease of which it is most frequently a result is scarlatina, in which it occurs as a consequence of the suppression of urine, from the uriniferous tubes becoming blocked up by epithelial scales in the nephritis which so often follows that disease. When the kidneys begin to act again, the effusion is soon absorbed. It never exists idiopathically.

The subjective symptoms of hydropericardium are



such as we might expect to find from obstructed circulation and respiration, and may be entirely wanting, of moderate degree, or so severe as to compel the patient to sit up constantly for fear of suffocation.

The physical signs are the same as those of serous effusion in pericarditis, with the exception that in the latter affection we may have *fremitus* and friction murmur, while in hydropericardium these signs are *never present*, there being a total absence of lymph.

According as the effusion is great or small in quantity, the impulse is either faint or entirely lost, particularly when lying down upon the back. In quite young subjects, the chest wall may bulge decidedly forward in the precordial region, and the intercostal spaces there be obliterated. There is increased breadth of dullness both to the right and to the left. If the effusion extends to the pleura, or if there be infiltration into the substance of the lung, producing dullness there, the value of dullness as a symptom is diminished. Auscultation shows the heart sound to be distinct but greatly enfeebled.

*Prognosis.*—The prognosis varies according to the nature of the disease with which it is associated. For example, when it occurs as a consequence of chronic heart disease, it is generally a signal that death is near, while that which follows scarlatina is not generally especially dangerous.

*Treatment.*—Arsenicum, Apis and Digitalis, according to the symptoms found in the materia medica, are the most effectual remedies we have for this condition.



In making choice, we should not lose sight of the nature of the cause or accompanying disease.

*Apis mel.*—Absence of thirst with scanty urination; sleeplessness; stinging burning pains in different parts of the body; dyspnœa, sensation as if he would never breathe again; must sit upright; especially useful after scarlatina, but very valuable also in a great many cases of general dropsy.

*Apocynum can.*—Can hardly speak for want of breath; great dyspnœa, wheezing breathing, and cough; heart's action scarcely perceptible; slow pulse, pulse small and irregular; face bloated and anxious looking; cannot lie down; general dropsy, urine scanty.

*Arsenicum alb.*—Inexpressible anguish and restlessness; no ease in any position; hydropericardium after scarlatina; dropsy consequent upon Bright's disease; anasarca, ascites; skin of face looks pale, earthy and greenish; great debility and prostration; tongue dry; great thirst, but drinks only a little at a time; respiration short and rapid; skin cool, burning inside.

*Digitalis.*—Faintness or sinking at the stomach, as if life was becoming extinct; symptoms of cardiac debility, as blue lips, rapid and difficult respiration, palpitation, weak, irregular and intermittent pulse; urine scanty, of high specific gravity, brick-dust sediment; general dropsy from heart failure; a sudden sensation in the chest as though the heart stood still.

*Kalicarb.*—With mitral insufficiency; heart's action tumultuous or weak, irregular or intermittent; pal-

pitation in spells, taking his breath; bag-like swelling between the upper lids and eye-brows.

*Lycopodium*.—Palpitation of the heart, in the evening in bed; acceleration of the pulse with coldness of the face and feet; shortness of breath; sensation of repletion in the stomach even after eating ever so little; feels about the hypochondria as though a hoop were drawn tightly round; red sand in the urine; oedema of the lower extremities.

(For other remedies see treatment of pericarditis.)

*Pneumopericardium*.—The patient is said to have pneumopericardium when the pericardial sac contains air or gas. It is rarely met with. It may perhaps be considered as an established fact that the contained gases have in all cases found their way into the pericardium by some opening, either as the result of wounds, or from abscesses which have broken into it from some neighboring organ, or which, originating within the pericardium, have broken outward. It has also been produced by ulceration of the stomach extending upwards. Pericarditis, unless already present, follows its admission. The accompanying effusion is nearly always purulent.

The subjective symptoms are not characteristic. We generally have, however, the symptoms of obstructed circulation, as dyspnoea, and a general cyanotic appearance.

The physical signs are very marked and scarcely mistakable. In cases of a very large quantity of gas,

with yielding chest walls, the precordium may even bulge forward. The most prominent among these signs is tympanites. The contained air being lightest, occupies the higher portion of the cavity, so that if the patient is lying upon the back, the entire precordium will be tympanitic, while in the upright position, the tympanites is only in the upper portion, above the base of the heart. The normal heart sounds, as also murmurs, if any be present, have a loud metallic ring. In some cases metallic tinkling may be heard, the result of the dropping of liquid effusion.

The prognosis of pneumopericardium is never favorable. Those cases resulting from wounds are least dangerous.

*Treatment.*—The physician should keep down the inflammation and preserve the vitality to the best of his ability by the administration of the properly selected Homœopathic remedies. (See treatment of pericarditis.)

## CHAPTER VI.

### DISEASES OF THE WALLS OF THE HEART.

*Hypertrophy.*—We come now to the consideration of the affections to which the heart walls themselves are subject, and, as most important among these, as among all cardiac affections, is hypertrophy, it will receive the first attention.

Genuine cardiac hypertrophy is not, as some have supposed, due to increase of connective tissue, but rather to the augmentation of the muscular fibre itself. Pathologists are not agreed as to whether this takes place by enlargement of the individual muscular fibres, or by an increase in their number. Most probably, however, both of these changes are going on at the same time. The increased growth takes place then, not by additional layers upon the surface of the heart, as the language of some writers might lead us to suppose, (Niemeyer and Fothergill both speak of compensatory hypertrophy surrounding dilatation,) but by an intermingling of new fibres with the old and the enlargement of those already existing.

Hypertrophy may be general, affecting the whole heart or partial, affecting the walls of only one or more of its chambers. It may be found of all degrees, from that which is almost imperceptible, to an increase of



heart whose normal weight is but eight or ten ounces to over six times its natural size. Hope saw one which weighed forty-two ounces, and Stokes exhibited one before the Pathological Society, the weight of which was sixty-six ounces; but such weights as these are of course extremely rare. The hypertrophied heart is somewhat darker in color than the normal, the greater the hypertrophy the darker being the hue.

This affection was formerly considered as a disease of itself, and treated as such with the idea of its removal, depletions and starvation entering largely into the treatment, but close study and observation have demonstrated the fact that it is, instead, only a compensatory effort of nature by which she seeks to overcome some obstruction to the circulation, to prevent undue distention of any of the chambers of the heart, or to repair or prevent injury to its walls, and that the greater the amount of vitality the patient possesses, the more readily does the heart take on hypertrophy when exposed to influences which would under other circumstances produce dilatation. It takes on this increase in dimensions and power, in the same manner as the arm of the blacksmith becomes more muscular than that of him whose occupation does not require so great an outlay of strength; but just how it is done is not known. Rutherford and others claim that the sensation of distention, of disability, is received by the cardiac ganglia; that through these the vaso-inhibitory nerves are excited to action, that this permits the vessels of the coronary circulation to open wider and re-

ceive a greater volume of blood, and that thus the heart walls receive the food required for an additional growth.

Hypertrophy may, for convenience, be divided into the simple and the excentric. In the simple form, the walls are merely thickened without alterations in the size of the cavity, while in the excentric form, not only are the walls thickened, but the cavity is more or less increased in size. This is hypertrophy and dilatation combined, and is of more frequent occurrence than simple hypertrophy. By some authors it is called hypertrophy with dilatation when the hypertrophy predominates, and dilatation with hypertrophy when the dilatation predominates. The heart may also be enlarged by increase in the size of its cavities and thinning of its walls, there being no increase of tissue. This is simple dilatation, and will be considered hereafter.

The left side is more frequently hypertrophied than the right. The amount of hypertrophy in any given case may be approximately arrived at on autopsy, when we remember that the average normal diameter of the wall of the left ventricle is about half an inch, of the right, from one-third to one-half as great, of the auricles about one line, the right being a little thicker than the left.

*Etiology.*—When the cause of hypertrophy presents itself suddenly, dilatation will be likely to be its first effect, to be followed or not by compensatory hypertrophy, which may be complete or incomplete, but

when it comes on gradually we may have simple hypertrophy from the start, or, if the vitality be deficient, the excentric form will most likely result, either hypertrophy or dilatation predominating, according to the vitality of the patient and the character of the cause. These causes may be divided into:

1. Obstructed circulation.
2. Distention under increased pressure.
3. Habitual excitement of the heart.
- I. Any obstruction to the blood stream.

*a.* First and among the most important, stands stenosis, or narrowing of the aortic orifice, generally the result of chronic endarteritis deformans, otherwise called atheroma. This species of obstruction is of slow growth, the heart has abundant time to prepare for the changing circumstances, and the hypertrophy is therefore generally pure.

*b.* Next to this in importance perhaps is aortic atheroma without stenosis of the orifice, this chronic inflammation and thickening of the lining membrane, destroying the elasticity of the aorta, either in its entire length, or in patches only, forming irregularities in the diameter of the vessel, but in either case making an obstacle in the way of the onward flow of blood, instead of assisting in the circulation. This atheroma is usually the result of strain, which may be produced in many different ways. One, in particular is by Bright's disease of the kidneys, where the systemic arterioles are contracted and the heart hypertrophied, the abnormal pressure at both extremities of



the blood stream at the same time, overtaxing the aorta and larger arteries. Atheroma of the pulmonary artery and its branches may be a cause of hypertrophy in the right ventricle.

*c.* Aortic aneurism is frequently mentioned as a cause of hypertrophy, but I believe that as yet no case has been published which could not be more rationally accounted for in some other manner. Indeed, the tendency should rather be towards dilatation and degeneration, as the aortic recoil must be imperfect and the coronary arteries incompletely filled.

*d.* Another cause is found in any occupation which calls for excessive labor with the arms, as in strikers at the anvil, who use their utmost strength in striking rapidly and with force while the iron is hot, at the same time maintaining a constrained position in order to strike the right place with precision; or, in those working in coal mines where the seam is not thick enough to permit them to stand erect. The result of this species of labor is, that the muscles, acting always towards the body, hasten the circulation in the veins, filling the heart's cavities to excess, and calling for extra exertion on its part, at the same time that by the constrained position, certain of the muscles are rigidly contracted across the arteries, obstructing the blood stream. The same cause also, by straining the coats of the larger vessels, produces atheroma in them, which in its turn becomes a cause of cardiac hypertrophy. When any kind of labor becomes a cause, we frequently have more or less dilatation, until compen-



satory hypertrophy is established, so that, in the earlier stages, the patient is often afflicted with palpitation, which afterwards ceases as the heart walls become strengthened by the compensatory growth. Sometimes, indeed, the compensation is never set up, and the patient is rendered an invalid for life, with a dilated heart.

*e.* The heart may be so placed as not only itself to labor at a disadvantage, but to create an angle in the pulmonary artery and aorta, partially cutting off the blood stream in the vessels. Under such circumstances nature makes an effort to create an equilibrium of forces by a compensatory growth, and thus we have another cause of hypertrophy; but not only is hypertrophy so produced, but the proper nourishment of the heart is interfered with, and we have, in time, dilatation and degeneration added.

One of the disturbing elements may be a pleuritic effusion, which is in many cases so profuse as to crowd the heart entirely out of its proper position. A like result is seen in rachitis and other causes of malformation of the thorax. In a patient of mine, the sternum was depressed so that in its middle portion, it was not more than an inch and a half from the spinal column, and the heart was situated entirely in the left half of the thorax. Rachitic persons frequently die from imperfect circulation and cardiac degeneration, brought about by the displacement thus produced, for the hypertrophy under such circumstances is almost cer-

tain, sooner or later, to degenerate into dilatation and decay of the heart walls.

*f.* Tumors may press upon the aorta so as to constrict it and interfere with the circulation, and thus become a cause of left side hypertrophy.

*g.* Common causes of enlargement of the right ventricle are obstructions to the circulation in the pulmonary artery, as from tumors, aortic aneurism pressing upon it, congenital narrowing, or disease of the left side of the heart; also anything which prevents the free flow of blood through the lungs, as bronchitis, emphysema, interstitial pneumonia, hepatization and pleuritic effusions.

*h.* A frequent cause of hypertrophy, especially in the aged, is chronic Bright's disease of the kidney. A portion of the kidney becoming, by chronic inflammation, unfitted to perform its functions, the blood is imperfectly depurated, and the retained urine salts cause, by their irritant action, spasmodic contraction of the minute arteries of the entire systemic circulation, with consequent deficiency of arterial blood in the periphery, and the characteristic coldness of skin, especially of the extremities. This contraction of the arterioles being an obstruction to the circulation, the left ventricle becomes hypertrophied, to meet the increased resisting force. Increased power of the ventricular contraction produces strain upon the arterioles in turn, and their coats become thickened and their caliber permanently lessened, while the cardiac hypertrophy increases. We now have an

unusual force acting upon the blood column at the ventricular end of the circulation, and another resisting at the peripheral end, and between these two, the aorta and the larger arteries become affected with strain and consequent atheroma. When the aorta becomes thus diseased, its recoil is less vigorous, the coronary arteries are consequently imperfectly filled, the heart is less perfectly nourished, and we have dilatation, to be followed in time by fatty degeneration, entire failure of the circulation, and death.

The order of sequence of these various changes may be briefly stated as follows:

First, kidney disease; second, impure blood; third, spasmodic contraction of the arterioles; fourth, hypertrophy of left ventricle; fifth, consequent strain on the arterioles, with thickening of their coats to resist this strain; sixth, more hypertrophy; seventh, atheroma of aorta, with diminished elasticity and imperfect recoil; eighth, ventricular dilatation, with systole enfeebled; ninth, diminished recoil of aorta, and further heart failure in consequence, with the effect of an obstructed circulation on the diseased kidney; tenth, fatty degeneration, dropsy from heart failure, general decay, and death.

II. Distension under increased pressure. This condition obtains in mitral and aortic regurgitation, and especially the latter. The resulting hypertrophy is generally of the excentric variety. At first, if the lesion be of sudden growth, we have dilatation, then



compensatory hypertrophy with dilatation, to give way later to nearly or quite pure dilatation.

Mitral regurgitation is the more frequent, and operates as a cause of hypertrophy in both ventricles, and of dilatation in the auricles; first, however, the left auricle, which may become also slightly hypertrophied.

The enlargement of the right ventricle in this case results from the over-crowding of the pulmonary circulation, which proves an obstacle in the way of the complete expulsion of its contents, and the pulmonary circulation is over crowded by the regurgitation through the mitral orifice, just as a portion of the water sinks back into the well when the lower valve of the pump is imperfect.

The left ventricle is enlarged by too great distention during its diastole, the blood which has regurgitated into the auricle added, to that which comes in from the pulmonary circulation, increasing the volume of that fluid to such an extent as to cause the ventricular walls to give way when the auricle contracts upon its contents, and we have ventricular dilatation, which soon becomes compensated by hypertrophy.

Aortic regurgitation is a not very infrequent phenomenon, and results from incapacity of the aortic semilunar valves. After being expelled from the left ventricle into the aorta, a part of the blood is forced backwards again by the recoil of that vessel, and a portion of it, from the fact that the valves do not close properly, is permitted to return into the ven-



tricle. This it does with great force, and, added to the stream coming in from the auricle during diastole, over-fills the ventricle and makes a pressure upon its now relaxed walls, which results in dilatation. Again, as the blood regurgitates during aortic recoil, instead of passing into the coronary arteries as it should, the latter vessels are but imperfectly filled, the heart walls do not receive the amount of blood which they require for their nourishment, they become debilitated, and the dilatation is increased. Nevertheless, nature, through the nervous system, makes an extra effort under these trying circumstances, and perhaps by making a better use of the blood supply, would seem in many cases to have overcome the dilatation by a thickening of the ventricular walls, but this cannot last long, and soon again dilatation comes to predominate. Soon too, the mitral valve becomes incompetent, the pulmonary circulation is obstructed, and the right side becomes also enlarged.

As the regurgitant current in aortic insufficiency possesses greater force than the forward current from the left auricle in case of mitral regurgitation, the ventricle is distended to a greater extent, the resisting power required is correspondingly greater, and the hypertrophy proportionately more marked. It is here, in fact, that we find the true *cor bovinum*.

### III. Habitual excitement of the heart.

In the condition known as irritable heart, resulting sometimes from long, hard marches, or from frequently repeated and long continued rapid motion of any

kind, a moderate degree of hypertrophy is a common accompaniment. The excessive cardiac action resulting from habitual and immoderate drinking is also an occasional cause.

Some pathologists claim that hypertrophy may result from temporary causes of debility in the heart walls themselves, and among these causes is mentioned exhaustion of the sympathetic from whatever cause, as excessive sexual intercourse, prolonged debauches, or immoderate smoking. It is asserted that the dilatation thus induced is compensated by hypertrophy after the cause is removed, provided that the vitality is good enough, and that appropriate treatment be instituted, but this is at least doubtful. It seems to me more natural to suppose that with the removal of the cause of the dilatation, and the administration of the proper remedy, the heart will resume its normal dimensions. A familiar comparison might be found in the uvula, which, during an acute catarrh, becomes elongated, flabby, and pendent, and when recovery takes place, resumes its natural character and shape by a renewal of strength and vigor and not by any addition to its substance.

Hypertrophy is more frequently found in males than in females, the advantage being as two to one in favor of that occurring in males. The cause of this difference, is most likely to be found in the habits of the male sex, which expose them to greater vicissitudes of climate, to more prolonged and extreme cold, and to more violent physical labor.

Hypertrophy and dilatation are so intimately connected in their etiology as to make any attempt to consider them separately, exceedingly injudicious. Nearly all cases of hypertrophy have been in the beginning, cases of more or less pure dilatation, while the vast majority must ultimately end in the latter condition.

Some cases of dilatation, from the very nature of their cause, can never result in hypertrophy, but must continue such, unless cured by the proper means, such cases being produced by any kind of chronic debility, as indigestion, phthisis pulmonalis, chronic diarrhœa, chronic uterine disease, etc. In some of these cases the cardiac disease may be the primary cause, when it must be treated as such by cardiac remedies, but in the majority of cases it is a secondary affection, the heart merely partaking of the general weakness, which must be treated in its own proper manner. This will be considered more fully elsewhere.

*Symptoms and signs of hypertrophy of the left ventricle.*—Hypertrophy is seldom discovered until dilatation has set in with it, from the fact that a pure hypertrophy is marked by no symptoms which attract the attention of the patient, and it is not until it has become complicated by dilatation, that we have palpitation, irregularity, and dyspnœa.\* These are the symptoms which drive the patient to his physician, the palpitation being the symptom which alarms him most.

\*The exception to this is where marked dilatation precedes hypertrophy.



Many people, physicians as well as laymen, imagine that this is an indication of hypertrophy, but such is not the case; it is, on the contrary, an evidence of its absence or deficiency. The greater the hypertrophy in proportion to the amount of dilatation, the less palpitation do we have. Palpitation, then, is a sign of weakness, and the same may be said of most of the other symptoms met with in cardiac enlargement.

The palpitation, irregularity and dyspnoea of eccentric hypertrophy then, are generally felt worse upon exertion, because then the heart is called upon to perform a labor beyond its strength.

*Physical Signs of Pure Hypertrophy.*—If we examine a case of simple hypertrophy by inspection, we shall see the apex beating at a small point below and to the left of its normal position, the degree of its displacement being dependent upon the amount of hypertrophy, it being sometimes found as low as the eighth intercostal space or even lower, and some distance to the left of the mammillary line.

Palpation discloses the apex, beating with much force over a small area, in contrast with the more diffused heaving found where there is considerable dilatation existing with the hypertrophy, and shows its position altered as above.

Percussion shows a greater extent of dullness, corresponding to the degree of enlargement, and discloses a heart not greatly increased in width, but much longer, the dullness extending some distance diagonally downwards and to the left.



On auscultation, the powerful heave of the hypertrophied heart is communicated to the ear by the stethoscope, particularly if the instrument be a wooden one.\* The first sound may not be quite so clear as the normal, and is heavier and more prolonged, the valve sound, although of course louder than in health, being partially concealed by the greater distance which it has to travel, while the muscular element is exaggerated.

On close examination it will be found to be reduplicated, the muscular element, which marks the beginning of systole, preceding the sound of valve tension, which marks its acme. One of the most marked features is the evidence of great power, usually without excitement. If the hypertrophy result from aortic regurgitation, the aortic element of the second sound is lacking, and we have in its place a right basic diastolic murmur. If it occur as a consequence of mitral insufficiency, the mitral element of the first sound is lacking, and we have in its place a systolic apex murmur, hence, when either of these murmurs manifests itself, it will be well to look for the signs of hypertrophy. If, however, a presystolic apex murmur alone is audible, hypertrophy will not be likely to be found, but rather the opposite condition of *diminution* of the size of the ventricle and thinning of its walls, from the diminished amount of blood which enters it through the narrowed orifice.

The pulse is an important aid in diagnosing a pure hypertrophy in the uncomplicated form, being large,

full, strong and sustained, so firm indeed as to raise the finger on each pulsation. It is, however, greatly modified by the co-existence of left side valvular disease.

*Physical signs of the excentric form.*—In proportion to the amount of dilatation present, the apex beats over a wider space than in pure hypertrophy, and this increased area is quite perceptible to the eye. The heaving impulse, in fact, is visible beyond the limits of the heart itself.

By the hand, this impulse is felt as not so powerful, but more violent, and more diffused according to the degree of dilatation, extending, frequently, far beyond the limits of the heart, and shaking the entire anterior chest wall. There is also double impulse, a secondary, lesser impulse immediately after the first, at the beginning of diastole, and caused, doubtless, by the rapid filling of the enlarged ventricle.

Percussion shows the area of dulness considerably increased in width and somewhat diminished in length, as compared with pure hypertrophy, the apex marking a more obtuse angle, and the border rounded so that deep dullness extends to the left, considerably beyond the apex beat.

On auscultation, the heart sounds are found more nearly normal than in simple hypertrophy, the valvular element of the first being more distinct, and not quite so much concealed by the thickened heart wall. There is, as in the simple form, reduplication of the first sound from separation of its two elements. The

second sound is not accented as it frequently is in pure hypertrophy. If the dilatation be considerable, irregularity, worse on exertion, is commonly present, with more or less palpitation.

*Auricles.*—Hypertrophy may be said never to exist in the auricles without dilatation, and most frequently the latter phenomenon is the leading feature; still, in many cases, there may be considerable thickening, as in mitral stenosis or even in regurgitation.

Physical signs are usually absent, particularly in the left auricle, as it is concealed behind the rest of the heart. The right auricle will be considered in the proper connection and is somewhat more favorably situated.

*Right side.*—While hypertrophy may be said to be characteristic of the left heart, dilatation may be considered equally so of the right. Right side enlargement may exist either with or without enlargement or other lesion of the left side, and is never to any considerable extent, if at all, dependent on left side changes as long as the mitral valve remains perfect. The other causes have been already enumerated but a repetition of them here will do no harm. Anything which obstructs the flow of blood through the pulmonary artery may be a cause, as a tumor or other objects pressing upon it. Obstruction to the circulation through the lungs is a most frequent cause, as from interstitial pneumonia, tubercular deposits, hepatization, and pleuritic effusion. Another prolific cause is any kind of exercise which calls for violent respiration, as



running, swimming, etc. The demands upon the right ventricle in these exercises, develop hypertrophy, and the more perfect is this, the better is the “wind” said to be, but in the course of time this hypertrophy gives way, the enlargement partakes more of the character of dilatation, and the subject of it grows short of breath, and is later affected with dyspnœa upon every little exertion.

*Physical signs.*—Enlargement of the right ventricle makes the inferior border of the heart almost horizontal, so that it seems to lie upon the diaphragm from near the apex to beyond the ensiform cartilage.

The heart beat is usually seen extending from the apex of the right ventricle to and even sometimes beyond the ensiform cartilage. The right ventricle being situated as it is, at the right and in front of the left, and being at the same time enlarged, frequently prevents the left ventricle from making any visible impression upon the chest walls, so that the apex appears to be beating in its normal position, although percussion may show it to be displaced considerably to the left, but never downwards. If, however, both ventricles are hypertrophied, of course we have the signs of the two combined, but each modifying the other.

The pulsation of the enlarged right ventricle can be felt by the hand, to which it does not, however, convey the impression of great power.

After the tricuspid valve gives way, the right auricle becomes considerably enlarged, and there may fre



quently be seen and felt a distinct pulsation about the third right rib.

The examination of the right side of the heart by percussion is not always an easy matter, but if we ascertain that the left ventricle is not enlarged, and that the apex is not displaced downwards, we have some negative evidence which may be of use to us, as it enables us to know that the increased action is on the right instead of the left side. We have the dullness of an enlarged right ventricle extending downwards and to the right beyond the normal limits, but to distinguish this from the dullness of its neighbor, the liver, is not always easy. This is aided, however, by the former rounding upwards and to the right of the ensiform cartilage, the enlargement extending, after the tricuspid valve gives way, as far as the border of the third right rib. Thus it extends not only downwards, but to the right of the limit of normal dullness,

Auscultation shows the first sound full and strong, and apparently produced near the surface. The tricuspid valve can be distinctly heard at the ensiform cartilage. We have also a distinctly accented second sound over the pulmonary artery, constituting a valuable diagnostic sign, showing increased recoil of that vessel, resulting from the more vigorous contraction of the powerful right ventricle, while the pulse is not in the least affected, except perhaps in frequency, and, in the later stages, after dilatation has come to predominate largely it may be slightly irregular. This freedom of the pulse from the influence of right

side hypertrophy, is a fact which should never be forgotten.

There is usually more or less cardiac dyspnœa. Very common attendants of this kind of lesion are the symptoms of the pulmonary disease which has been its cause. When the case has proceeded so far as to cause the tricuspid valve to give way from dilatation, or what is less common from valve disease, then does dilatation of the right auricle manifest itself, with great venous congestions, anasarca, etc., and the case passes rapidly forward to a fatal termination.

*Consequences of Hypertrophy.*—One of the consequences of hypertrophy, both simple and excentric, is cerebral apoplexy. This, however, is not likely to occur before the age of forty years, however great the hypertrophy may be, and as previous to this period, atheroma, by which the arterial coats are rendered brittle and easily ruptured, is rare, it may be considered as pretty evident that without this disease in the arteries, hypertrophy is incapable of producing a rupture of their coats, and consequent effusion. Atheroma of the cerebral arteries, then, may be taken as the predisposing, and left ventricular hypertrophy as the exciting cause.

Pulmonary apoplexy has usually been regarded as a result of right side hypertrophy, and this may be the true explanation of it, but Hayden\* refers it rather to the existence of a clot in the right ventricle and pulmonary artery, obstructing the supply of blood to

\*Diseases of the Heart and Aorta, pp. 517-18.

the lung. He claims that the engorgement and hæmorrhage are the result of influx from the veins, and paralysis of the capillaries.

Dropsy is a result, not of hypertrophy itself, but of the cardiac debility which follows it. That which occurs as a consequence of chronic kidney disease, and which may be found accompanying hypertrophy without dilatation, may be distinguished from cardiac dropsy by the puffiness under the eyes, and the absence of the symptoms of heart failure, as the feeble apex beat, the palpitation and irregularity on exertion, and the general venous engorgement.

The heart may be so enlarged by hypertrophy or dilatation, or both combined, as to produce dyspnœa by its pressure upon the lungs; or a hypertrophied heart may throw the blood into a dilated aorta with such force as to produce a like result by its pressure upon the lungs and bronchi, but cardiac dyspnœa, like cardiac dropsy, is usually the result, not of hypertrophy, but of the succeeding heart failure.

Another important consequence of hypertrophy is dilatation of the aorta, and, at the same time or subsequently, atheroma; and this atheroma may not be of the aorta alone, but of the entire arterial system. With aortic disease, as before stated, comes imperfect recoil, with imperfect filling of the coronary arteries, and consequent weakening of the heart walls, dilatation, and afterwards perhaps fatty degeneration, with the consequences of heart failure to the general system.

Fatty degeneration, then, often follows hypertrophy,



not as a direct result of that condition, but of the imperfect nutrition of the heart walls, most frequently consequent on atheroma.

*Simple dilatation.*—Dilatation may be divided into the primary and the consecutive.

Among the causes of primary dilatation will be found anæmia, the excessive use of tobacco, especially smoking, masturbation, sexual excess, softening of the heart (typhoid, fatty, or inflammatory) and over exertion with imperfect nutrition.

Consecutive dilatation is caused by anything which is capable of producing hypertrophy, and generally succeeds that condition.

It may also be divided into general and partial, the cases of the latter predominating in point of frequency.

General dilatation is always of constitutional origin.

Among the symptoms of a moderately well pronounced case of simple dilatation, are more or less of general debility, with mental and bodily languor. The pulse is feeble, quick, and sometimes intermittent, and this intermittency is not merely irregularity, but a complete cessation of the heart's action during the period of one or more pulsations. If this occurs particularly after exertion, it is a symptom of very serious import. The heart is for the time over-powered and waits during the time in which one or more contractions should take place, until it can gather strength for another effort. It is not an intermittency of the pulse alone, but also of the heart's action. The skin



and extremities are cold, slightly bluish, and easily chilled on the least exposure, with great disposition to catch cold. There is usually considerable dyspnœa, particularly on exertion, and slight teasing cough with but little expectoration. The appetite is generally deficient, and the digestion frequently disturbed.

In the later stages of a bad case, all the above symptoms will be found greatly aggravated. The cough is now worse, and attended with profuse serous or thin mucous expectoration, and the dyspnœa is so great as to prevent the patient from lying down. He has a somewhat jaundiced appearance. The signs of dropsy are everywhere visible, with cold skin and blue nose, lips and ears. The pulse is now slower, feebler, more intermittent, and more irregular, with much palpitation and vertigo, especially on rising up or on exertion. If, however, extensive fatty degeneration is present, no palpitation is felt, the heart being apparently incapable of making any unwonted exertion, or of being excited to any unusual action.

*Physical signs.*—Except in persons very thin in flesh, the impulse of a dilated heart cannot be seen, but when visible, the apex beat is scarcely distinguishable from that of the entire ventricular portion of the heart, the impulse appearing as a more diffused, feeble slap, visible only in the intercostal spaces, throughout a great portion of the cardiac region.

Palpation shows similar results, the impulse being felt, if at all, as very feeble, and widely diffused.

Percussion shows the area of precordial dullness

more extended than natural, and of a rounder form than in hypertrophy.

Auscultation displays the muscular element of the first sound as a sort of feeble, trembling motion, difficult to describe, but readily recognized by the practiced ear. The valvular element is most noticeable, and is clear and distinct, although much weaker than natural. It may occasionally be replaced by a systolic apex murmur from regurgitation through the mitral orifice, not the result of disease of the mitral valve, but of the giving way, at the acme of systole, of a portion of the ventricular wall to which one of the papillary muscles is attached, the flaps being thus drawn out of position through the agency of the tendinous cords, and prevented from completely closing the orifice. The second sound is usually feeble.

When dilatation proves fatal, it terminates in effusion into all the serous cavities, and death takes place from coma produced by the imperfect purifying of the blood. Hayden mentions another form of death; that by the formation of a clot in the right side of the heart and the pulmonary artery, from lack of vigor in the circulation. This condition may be diagnosed by the great *sense of suffocation*, the continued *violent struggling for breath*, although the air freely enters every part of the lung, the *tumultuous and irregular action of the heart*, and the *throbbing carotid and temporal arteries*. The suffocation proceeds not from deficiency of air in the lungs, but from a deficiency of the blood to be purified, the clot obstructing the

supply of venous blood from the right heart to the lungs.

*Prognosis of Cardiac Enlargement.*—In arriving at a correct prognosis of a case of cardiac enlargement, it will be necessary to take into consideration the patient's age, evident vitality, habits of life, and in particular, the condition of the heart itself, the nature of the lesion, its cause, and the amount of hypertrophy in proportion to the dilatation. If the compensation be perfect, there being no dilatation, the chances are that the patient may live for a considerable number of years in tolerable health and with his usual strength, but all things else being equal, in proportion to the amount of dilatation present will his career be shortened, and his ability to move about the world as do those in health be lessened. Hence, the more perfect compensation we can obtain, the better will it be for his welfare. When once pure hypertrophy has been established, it should be our endeavor, as far as possible, to preserve that condition to the extent of not allowing dilatation to be set up in its stead, for the chief danger to life from hypertrophy, is found in failure of the circulation from the dilatation which so generally follows it, in consequence of the hypertrophied left ventricle having broken down the arterial system by its powerful contractions, and thus prevented the aorta from recoiling with sufficient force to supply the coronary arteries with their due share of blood. Everything which increases the heart's action increases the violence of the ventricular contractions



and tends to hasten the breaking down of the arterial system. For this reason, the subject of cardiac hypertrophy, however pure that may be, and however strong and capable of physical exertion he may feel himself to be, should lead as quiet a life as is consistent with good health and vigorous digestion. If he passes to the opposite extreme, of leading too sedentary a life, and thus brings about a feeble condition of the digestive apparatus and nervous system, he will soon learn that he has hastened the very end he has sought to avoid. It will perhaps be well to remember that moderation is not incompatible with industry, although care should be taken in selecting the occupation.

Another danger from hypertrophy is that of cerebral apoplexy, the arterial system having first become diseased, but this danger is not great, the percentage of cases of heart disease in which apoplexy occurs being but little above that of healthy hearts.

As to dilatation, that which is the result of any cause capable of producing hypertrophy, is more certainly and speedily fatal than the primary form, while that which occurs after compensation has once been set up, being the beginning of the end of the series of cardiac changes, is far more serious than that occurring previous to hypertrophy, for in the latter case the dilatation may yet be compensated for, and a condition of health maintained perhaps for years. There is, however, one form which I have scarcely mentioned as yet, and that is the dilatation resulting from fatty degeneration, and which is much more dangerous than



any other form. Simple primary dilatation, however, while being a source of great discomfort, can yet scarcely be said to be an especially dangerous affection, provided proper care be taken and no excess committed.

While any given case of cardiac enlargement of whatever nature, be it one of hypertrophy or dilatation or of both combined, may not of itself prove fatal, yet it tends greatly to diminish the amount of vitality the patient possesses, and thus renders an attack of any other disease more dangerous.

Concerning the amount of exercise that may be taken by the subject of cardiac dilatation, I shall not say that a condition of perfect rest should be maintained, but rather that the strength of the patient may be taken as a pretty reliable guide. In many cases, and particularly those dependent upon indigestion, a considerable amount of active exercise may be taken with profit, provided that the case has not progressed too far, while in fatty degeneration, except perhaps in the earliest stages, it would perhaps be impossible to maintain too quiet an existence. Here palpitation might be considered of good omen as indicating that degeneration is not yet very far advanced.

*Treatment.*—Can *pure compensatory* hypertrophy be cured by medicine? This is a question not unfrequently asked, and I now answer most emphatically that in my opinion it can not, and for the reason that it is not a disease. By starvation and the use of depleting medicines, it may be and frequently has been

converted into dilatation, but this is a long stride towards a fatal termination, and the very thing above all others, to be avoided. If the cause of hypertrophy be removed, as for instance, the too violent exercise, the cardiac excitement, or the obstruction to the circulation, the natural tendency must be to a return of the heart-walls to their normal dimensions and condition, just as the muscles of the blacksmith's arm diminish in size when he exchanges his laborious occupation for one of a lighter character; provided at the same time that the hypertrophy has not existed so long as to induce changes in the arterial system, or in the valves, but unfortunately it is usually not discovered until these changes have been going on for some time. For this reason, and from the fact that in the vast majority of cases the cause is irremediable, the normal condition is seldom restored.

To the end that we may have a proper understanding as to what is required in the treatment of a case of hypertrophy, it becomes necessary for us to know what it is that we have to treat; what is disease and what is not; and what it is we have to fear as a cause of death or suffering to our patient. First then, as far as the heart itself is concerned, the essential disease element of enlargement is the dilatation, and this it is that must receive our attention with a view to reducing it, and restoring the integrity of the muscle of which the heart is composed.

But the dilatation is not the only thing to be considered, for hypertrophy, although not in itself a dis-

case, but a compensatory growth, an effort of nature to restore to the heart walls their lost strength and integrity, to overcome some obstruction to the circulation, or to enable the ventricle to meet the otherwise increased demands upon it, is not very rarely a cause of death. But how, it may be asked, can hypertrophy, if it be a healthy growth, ever be a source of injury to him who has it? My answer is, that it is not a cure but a temporary expedient; it is nature's palliative in the true sense, and must sooner or later break down the foundation upon which it is built, but as nature could in no other way overcome the obstruction or remove the dilatation, it is better that it should be so, than that the system should break down earlier from the primary disease. It is a palliative measure as the watery discharges from the bowels in an advanced case of venous engorgement are palliative. It is in some sense a removal of the consequence of disease, but unless in the mean time the cause be removed, these consequences must at some time reappear. Again, as already shown, it not only brings about its own destruction, but may be itself a cause of death.

Thus it will be seen, that although a healthy growth, and nature's own restorative, it is not altogether a thing to be desired, but when the alternative is given us, to have either hypertrophy or consecutive dilatation, we should by all means choose the former, for while dilatation is decay, hypertrophy is temporary restoration.

The old way of treating hypertrophy was to reduce



it by the means best intended to diminish muscular power, starvation, depletions, and depressing medicines being found most effective, but now even Old School physicians have learned better, and their works upon the diseases of the heart, caution the reader to avoid this destructive method, but certainly a Homœopathic physician would need no such admonition.

The first thing to be done, is to ascertain, if possible, the cause of the hypertrophy, and when found, no matter of what nature it may be, whether the habits of the patient, or disease in some other organ or organs, everything that is possible most be done to remove it, both medicine and hygiene being placed under contribution to that end.

Next, always remember that there can be no such thing as a direct Homœopathic remedy for pure compensatory hypertrophy as an individual disease, and that any medicine supposed to be such and administered for the purpose of its removal, must prove, as far as the heart is concerned, either injurious or inert, for Homœopathy applies to the cure of *disease* only.

Where then does medicine find its place, and what must be our aim in its use?

1. To remove the cause or causes.

2. To preserve or restore, by direct or indirect medication, the integrity of the heart walls, preventing or curing dilatation or decay, at the same time that we remove as far as is in our power, all unhealthy or extravagant action which may be the result of excitement but not of power.



3. To remove those diseases and symptoms of disease in the general system, which are the result of the cardiac lesion.

At the same time that medicines are given, it is necessary to make use of hygienic measures. If the patients habits are in a direct manner the cause of the lesion, those injurious habits must be broken up. He should endeavor so to live as to secure the very highest possible degree of nutrition, while he guards against exciting the heart to undue activity.

The medicines applicable to the removal of the cause of the enlargement are so different, according to the different circumstances and diseases upon which it may depend, that the attempt to specify in advance what is needed and under what circumstances, would likely be attended with failure. As to the removal of the consequences of the enlargement, and the treatment of the dilatation, something more explicit can be given.

While consecutive dilatation is only to a limited extent remediable, except by the substitution of hypertrophy, the simple primary form may be classed among the curable diseases. Even the dilatation of fatty degeneration, although not altogether curable, is susceptible of considerable temporary improvement.

The following remedies, among others, may be found useful in the various forms of enlargement.

*Aconite* is occasionally of use in removing the irritability sometimes present in hypertrophy, and which, with the augmented power of the ventricle, breaks down the arterial system, and might result in apo-

plexy. Among the indications are a full, quick, firm pulse, dry and hot skin, and thirst.

*Digitalis*, not to low, is also sometimes of benefit in cases of irritability, but its chief use will be found in cardiac debility, or dilatation, and it is here the remedy of the first importance. It acts better given low, and I should not scruple to use it even as low as the mother tincture, several drops at a dose, if it seems to be more efficacious, for the size and strength of the dose makes it no less Homœopathic to the case, and I understand the first principle of Homœopathy to be the similar remedy, and the second, the smallest dose that will cure with equal facility. With this medicine, many a moderate case of simple dilatation can be cured, and the beginning dilatation of many a hypertrophy be relieved. Even in fatty degeneration, the heart is often for the time strengthened by its use. We find it then a very potent agent in treating the dropsy and venous congestion resulting from cardiac debility.

Years ago Old School physicians considered this medicine to be what it is, a depressor and capable of paralyzing the heart's action; capable of producing in small doses a rapid and feeble pulse; in larger doses a small, soft and irregular pulse, but in either case, weakening the heart muscle, enfeebling its action; but having discovered that it *cures* these precise conditions, causing the heart to beat as slowly as natural only, and with strength and regularity, producing a full, regular, strong pulse, when before it was weak and irregular, increasing the amount of urine when before

it was almost entirely suppressed, they have arrived at the conclusion that it is a stimulant of cardiac action, and that its power to restore an enfeebled heart is dependent upon that stimulant property. And why have they thus explained its action? To avoid that confirmation of the Homœopathic law of cure which must attend any other explanation of it? They are probably honest in their convictions, but it seems to me that they are certainly very greatly influenced in this by preconceived opinions. They have depended for their knowledge of its action upon its effect in disease, instead of studying cases of poisoning and provings upon persons in health. Cases of poisoning with *Digitalis* show excessive cardiac debility, in proof of which, witness the slow, irregular, feeble, intermitting pulse, palpitation and even syncope on rising or other considerable change of position, and greatly diminished or almost totally suppressed urine. This diminution in the quantity of urine is in itself sufficient evidence of the cardiac debility which is always present in cases of poisoning with this drug. The increased volume of urine often seen, is only a curative effect, from the drug having been given for cardiac debility and consequent tendency to dropsy, and results from restoration of normal ventricular power, and consequent normal arterial tension on the kidneys, with a watery condition of the blood.

The symptoms given above are the leading, distinguishing features of its primary action. The secondary action, found when small doses are administered in



health, is characterized by rapid and feeble pulse, not by a rapid and strong pulse nor by a healthy, full, slow and strong pulse, like that which it produces in the cure of an enfeebled heart, so that there is no ground whatever for calling it a stimulant in any sense. This excited action with increased frequency is only a sort of demoralization, an excitement with debility.

The following symptoms indicate its use: Distressing dyspnœa; suffocative, painful constriction of the chest; dull uneasiness in various parts of the region of the heart; a sudden sensation as though the heart stood still, with great anxiety; action of the heart feeble and constantly accompanied by palpitations; the heart's action has lost its force, its beats are more frequent, and intermittent and sometimes irregular; scarcely perceptible beat of the heart; pulse small, feeble, and irregular or intermittent; urine scanty and with brick-dust sediment; anasarca; great prostration, fainting on the slightest movement, even lifting the arms; coldness of the limbs and body; faintness or sinking at the stomach as if life were becoming extinct.

See Hughes' Pharmacodynamics, Hempel's *Materia Medica*, Allen's *Encyclopædia*, etc.

*Cactus* is frequently useful, particularly in dilatation resulting from pericarditis.

*Symptoms.*—Palpitation of the heart, continuous, day and night, worse when walking and at night when



lying on the left side; the palpitation consists of small irregular beats, with necessity for deep inspiration; very acute pain, and such painful stitches in the heart as to cause him to weep and cry out loudly, with obstruction of the respiration; sense of constriction in the heart as if an iron band prevented its normal movement; general sensation of constriction in throat, chest, heart, bladder, rectum.

*Lachesis*.—Palpitation with fainting and anxiety; palpitation and choking, from the slightest exertion; constrictive sensation in region of heart; irregularity of heart-beats; cannot bear the slightest pressure over the part; all the symptoms worse after sleep. Especially useful in that form of dilatation resulting from debility and derangement of the nervous system at the climacteric. Hot flashes in various parts.

*Lycopus*, although a new remedy, and as yet but little used, bids fair to become one of our most valuable remedies in dilatation. Its effect is marked by weakness and irregularity of cardiac action, and very feeble, irregular pulse.

*Nux vomica* not only promotes vigorous digestion when Homœopathically indicated by the condition of that function, but exerts a very decided influence over the heart itself. It has many times shown its efficacy in dilatation as well as in nervous palpitation, not only palliating, but often permanently curing those affections. The patient is worse after a full meal, in the morning on waking, after loss of sleep, and in cold, damp weather. There are also dull headache, drowsi-

ness, loss of appetite, sour stomach, gastralgia, and constipation.

*Spigelia* is also a valuable remedy in dilatation. It has violent palpitation, with or without painful stitches in the region of the heart; worse when bending the chest forward, on rising up, on raising the arms, and in the afternoon. Violent vertigo frequently accompanies the palpitation.

*Spongia* should never be forgotten when the pulmonary symptoms are prominent, especially if the patient suddenly starts up from sleep at night, with great fright, and fear of suffocation. He cannot lie down in the after part of the night in consequence of the great dyspnœa. This symptom I have seen verified in my own practice, and this from the 200th potency, a single dose only being given before complete relief was obtained. Of course the relief could not be permanent, for it was a case that was very near the grave, but it would last for some days and then the distressing symptom would return, when another dose would dispel it as if by magic. This was repeated several times, always with the same result. The case was one of mitral disease, but the *Spongia* would have been equally applicable in dilatation from other causes. It is particularly indicated in people of light, nervous organization, with fair skin, light hair and blue eyes.

*Ignatia* might be beneficial in dilatation the result of grief, especially in women.

*Plumbum*, being capable of creating a tubular nephritis of a lasting character, is Homœopathic to

Bright's disease, and when the kidney is not too far gone, may restore to health at least a portion of the part involved. Most likely the cases of left ventricular hypertrophy which it is supposed to have created, have been secondary to kidney disease of which it was the cause, as we see in chronic Bright's disease, the imperfectly depurated blood, exciting spasm in the arterioles, and that spasm, by obstructing the blood current, producing hypertrophy, the process being similar in the two cases. If the case be taken in time, before the arteries and arterioles have been seriously injured, the removal or partial removal of the kidney disease, by rendering these organs more capable of purifying the blood, and diminishing the consequent spasm, may exert a curative influence over the hypertrophy, the demands upon the ventricle being diminished in proportion to the degree of restoration of the arterioles to their normal calibre.

Any other remedy which could restore the diseased kidney to health, and the performance of its functions, would of course have over hypertrophy, which is consequent upon renal disease, an equally beneficial influence to that possessed by Plumbum; but it is not my purpose here to give instructions for the treatment of Bright's disease.

In addition to those already mentioned, we have the following remedies from which to select:

*Amyl nit.*—Flushing of the face; feeling of constriction in throat, extending to chest; precordial anxiety; a very marked beating of the heart and



carotids; cardiac oppression, and tumultuous action of the heart; pain and constriction around the heart; increased frequency of cardiac pulse; pulse rapid and jerking; sphygmographic pulse, curve ends abruptly in a very sudden downfall; general relaxed, weak feeling all over the body.

*Apis mel.*—Great feeling of suffocation; it seems as if she could no longer survive, for want of air: dyspnœa, it seems impossible to breathe; stitches in the left side of the chest; sudden attack of acute pain just below the heart, soon extending diagonally towards the right chest. Blowing sound with the diastole; great oppression of the heart's action; pulse generally slower than normal, weak, and often almost imperceptible. General dropsy; absence of thirst with scanty urination.

*Arnica.*—Pain in the heart as if it were squeezed together, or as if it got a shock; stitches in the cardiac region; stitches in the heart from left to right; the beating of the heart is more like a jerking; pulse slow and full, or feeble, hurried and irregular; bruised feeling in the region of the heart; hypertrophy of heart from any kind of over exertion, as running or rowing. Fatty heart. Bayes speaks of having frequently cured hypertrophy, resulting from over exertion in young men, with this remedy.

*Arsenicum.*—Oppression of the chest when walking fast; palpitation of the heart; violent palpitation of the heart with small irregular pulse; irregular palpitation of heart, but so violent that he imagines he



hears it, accompanied with anguish; palpitation with tremulous weakness after stool; he has to lie down; irritable heart; quick, weak and irregular pulse, sometimes wholly imperceptible; great debility; dropsy. Most frequently indicated in the later stages.

*Aurum met.*—Frequent deep breathing; severe dyspnoea; excessive dyspnoea with difficulty of breathing at night; asthma from congestion to the chest, (heart failure;) great oppression of chest at night when walking in the open air; suffocative fits; with spasmodic constriction of the heart; face bluish red; palpitation, falls down unconscious; frequent attacks of anguish about the heart, and tremulous fearfulness; drawing and cutting pains at the heart; melancholy, suicidal mania; after mercury. Should be most useful in dilatation and the eccentric form of hypertrophy, when resulting from valvular lesions.

*Bromium.*—This remedy has been recommended for hypertrophy, occurring in young and growing subjects. It has: cutting pains running upwards in heart disease; violent palpitation when walking; also in evening, so that one cannot lie on left side; pulse somewhat accelerated, full and strong.

*Collinsonia*—Hæmorrhoids and constipation are the most prominent symptoms, (Burt.) Most useful in dilatation.

*Ferrum.*—Useful in some cases of simple dilatation in which the anæmia is very marked. Palpitations better when walking slowly; pulse sometimes accelerated, but generally slower than the normal; face

flushed and burning; hammering and beating in the head so that she sometimes has to lie down in bed.

*Kali carb.*—Heart's beat intermits; heart's action weak; tumultuous or irregular; stitches about the heart and through to scapula; palpitation in spells, taking his breath; throbbing and palpitation of the heart on the least exertion. Insufficiency of the mitral valves; pulse unequal, irregular, or intermitting; pulse slow and weak; bag-like swelling between upper eyelids and eyebrows.

*Kalmia lat.*—Oppressed breathing with palpitation of the heart, and anxiety; palpitation of the heart on walking slowly up stairs; pulse very feeble, or irregular; hypertrophy consequent on mitral disease, after rheumatism; wandering rheumatic pains in cardiac region.

*Lilium tig.*—Sharp, quick pain in left side of chest, with fluttering of the heart; constant feeling of load or weight in left chest; dull, heavy or pressive pain in region of heart; intermittency of heart's action; pulse small and weak; irritability, nervous palpitation; *amelioration by lying on the left side.* Likely to prove useful in simple dilatation.

*Lithium carb.*—Pain in the heart; worse in the morning after rising; sometimes a sudden jerk or shock in the heart; pain in the heart before and during micturition. Hypertrophy from valvular deficiencies, especially of the aortic valves, in persons of a gouty diathesis.

*Lycopodium.*—Useful in some cases of simple dila-

tation; palpitation, nearly every evening, in bed, with great fullness of stomach and abdomen, from flatulence; feeling of fullness after eating ever so little; cannot bear anything tight around the body; gurgling of wind in upper abdomen under the apex.

*Naja trip.*—Feeling of depression and lowness about the heart; great pain about the heart; fluttering about the heart with headache; pulse rapid and irregular in volume, but regular in rhythm; or, pulse very slow and irregular, both in volume and rhythm; cannot lie on left side.

*Tabacum.*—Paroxysms of præcordial anguish, with palpitation and pain between the shoulders, worse at night; violent palpitation, worse lying on left side; heart's action very feeble; pulse generally either too fast or too slow, and small, weak, and often irregular or intermittent; cold hands and feet.

*False Hypertrophy.*—We have in addition to the cardiac enlargement already considered, an enlargement by an abnormal development of connective tissue. This has been aptly termed false hypertrophy in contra-distinction to the true, which is enlargement by increase in the number and size of the muscular fibres. It is a rare disease and but little known, and hence difficult to diagnose. By it the heart may attain to an enormous size, but as the muscular fibre is not increased, but on the contrary frequently diminished by pressure of the increased connective tissue, we should have as signs of its presence, increased area of dullness on percussion,



without either hyper-activity, or the evidence of unusual power which is manifested in true hypertrophy.

*Etiology.*—Various theories have been advocated as to its mode of origin, some claiming that it is the result of venous congestion of the heart walls from defective circulation in the coronary veins. This is perhaps the most plausible theory, but still it would be difficult to say to a certainty whether this venous engorgement stands in the relation of cause or effect. On the other hand, it has been supposed by some to result from interstitial inflammation.

It can hardly be considered a dangerous affection unless the development be so great as to produce heart failure from destruction of the muscular fibres. As it is next to impossible to diagnose it, and as it is almost entirely devoid of symptoms, it would be difficult to write a section on the treatment. If any symptoms should arise, they should be met by a remedy selected in accordance with their characteristics. (See treatment of Hypertrophy and of Pericarditis.) Hayden, treating of this subject under the head of fibroid transformation, claims that it is the result of inflammation and that it can be arrested in its progress, if taken in the early stages.

*Cardiac atrophy* is a diminution or shrinkage of the muscle of which the heart is composed. It may be divided into 1. Simple atrophy, with thinning of the walls and normal or diminished size of the cavities. 2. Eccentric atrophy, with thinning of the walls and enlargement of the cavities by dilatation.



The organ exhibits, on post mortem, a somewhat paler color than natural, and is generally firm, and without a change of structure. If, however, fatty degeneration be present, it is more easily torn. In another and unusual form, it is of a reddish brown color, from deposit of pigment granules.

*Etiology.*—Atrophy is sometimes produced by the constant pressure of long standing effusions. Other causes are adhesion of the pericardium in such a way as to constrict the coronary arteries, calcification of the pericardium and of the plastic effusion of pericarditis, and general marasmus; but what are much the most frequently responsible are cancer and tuberculosis.

*Diagnosis.*—Inspection and palpation usually fail to detect the apex beat, while in many cases not yet complicated by dilatation, percussion shows the area of dullness somewhat diminished. The sounds are usually quite distinct and regular, though not so loud as in health. If degeneration be present, they may be all but imperceptible.

The pulse is usually regular, quick and feeble, and if softening be present, may be altogether lost. With softening also, if still palpable, it will be found *very* feeble, irregular and intermittent, and there will be occasional attacks of syncope.

There is in most cases more or less palpitation. General debility, vertigo, coldness of the extremities and shortness of breath are also present.

The *prognosis* is generally unfavorable, but worst of

all in those cases dependent on cancer and tuberculosis.

*Treatment.*—We shall find our best results from treatment in those cases which result from pressure of the effused fluid within the pericardium. (For such cases, consult the treatment for pericarditis and hydro-pericardium.) In those cases resulting from calcification of the pericardium, and constriction of the coronary arteries after pericarditis, as also those which have their origin in cancer, or met with in the advanced stages of tuberculosis, of course but little can be done; while those which are so often found in typhoid conditions, generally recover of themselves with the recovery of the general health.

For the treatment of those cases which result from general debility, as well as all those which are complicated with dilatation, see the treatment of hypertrophy and dilatation. Of the remedies therein mentioned, I would recommend more especially; *Ars.*, *Collin.*, *Dig.*, *Fer.*, *Lyc.*, and *Nux v.* In addition to these, the following are likely to be found useful:

*China*; after loss of animal fluids; ringing in the ears; bitter taste in the mouth, tongue coated white or yellow; empty eructations; tympanitic distention of the abdomen; diarrhœa of undigested food; palpitation of heart with intermittent pulse; excessive sensitiveness of entire nervous system; skin feels sore all over.

*Phosphorus.*—Tall slender people with sanguine temperament; with pulmonary affections; after ex-

hausting diarrhœa; after sexual excesses; after typhoid fevers; premature old age; sensation of weakness and emptiness in the abdomen; attacks of sudden blindness; anxiety about the heart with nausea and a peculiar sensation of hunger; systolic bellows murmur at base of heart, from anæmia; violent palpitation; pulse rapid and weak; weakness in all the limbs.

*Sulphur*.—Burning heat in the top of the head; after suppressed eruption, or itching of the skin aggravated by heat and relieved by scratching; alternating diarrhœa and constipation; hæmorrhoids; burning heat of the palms of the hands and soles of the feet; short stitches in the precordial region; palpitation at night in bed.

*Myocarditis*.—Inflammation of the muscular walls of the heart may be either acute or chronic. The whole heart is seldom involved, the inflammation usually confining itself to a single portion, most frequently the left ventricle. The muscular fibre alone, or the connective tissue alone, or both, may be affected. It is doubtful if it ever exists alone, either endocarditis or pericarditis being always found with it. Hayden says that it may be accepted as a rule that myocarditis is to some extent present in every case of endocarditis or pericarditis; usually however, in these cases, the myocarditis is so limited in extent as scarcely to be worthy of the name. The disease may extend only a little distance from the surface, or the entire thickness of the walls may be involved. It is most frequently



found in early life, and oftener in males than in females.

*Etiology.*—The most frequent cause is rheumatism. It may then be either acute or chronic, and affect a considerable portion of the heart wall, especially in the chronic form, or be confined to a limited area, as one of the papillary muscles, which is likely to contract so as to interfere with the closing of the valves. Rheumatic myocarditis seldom results in abscess.

Next to rheumatism may be placed puerperal, relapsing, and typhus fevers. Myocarditis thus occurring is acute, and is likely to end in abscess, which may break into the chambers of the heart, or into the pericardium.

It may result also from wounds, or from an embolus becoming lodged in the coronary circulation.

*Diagnosis.*—While a positive diagnosis is always difficult, it is by no means impossible. The most valuable diagnostic symptom is the extremely rapid and feeble pulse, so rapid often, that it cannot be counted—with irregularity and intermittency. In addition to this, there are fluttering and irregular action of the heart with occasional intermissions, feeble or suppressed impulse even when the patient is in the prone position, sounds of the heart extremely feeble and irregular, skin pale and cold, gasping respiration, with great desire for air, collapse of features, pain and great oppression in the precordial region, and symptoms of delirium.

It is distinguished from pericardial effusion by the



fact that in the latter there is increased area of percussion dullness, the impulse can generally be felt when the patient is lying in the prone position, and the pulse is not so rapid.

It might be confounded with thrombosis of the pulmonary artery, but in the latter, the pulse, although rapid and very feeble, is more regular, the carotids throb violently while in myocarditis they do not, and the cardiac impulse is violent while in myocarditis it is very feeble or entirely imperceptible.

*Prognosis.*—This is different in the two forms of the affection, the suppurative form being almost necessarily fatal, although cases have been known in which the pus became encysted in connective tissue, and the patients were afterwards restored to health. In most cases death would result from paralysis of the heart from inflammation, before the formation of pus. The rheumatic form is far less serious, the danger depending largely upon the extent of the inflammation. Here, too, the greatest thing to be dreaded is paralysis of the heart walls, but aside from this, the part inflamed is apt afterwards to undergo fatty degeneration.

*Treatment.*—In the suppurative form, no treatment is likely to be of any avail. In the rheumatic form, I would simply suggest that Aconite, a few drops of the first decimal dilution or mother tincture, in water, should not be forgotten. (For further information, consult the articles on pericarditis and endocarditis, and study the *materia medica*.)

Fatty disease of the heart is of two kinds, fatty degeneration, and fatty deposit or fatty infiltration

*Fatty Degeneration.*—By degeneration is meant a disease entirely distinct from fatty infiltration, the latter disease, if such it can be called, attacking only the areolar tissue, and affecting the muscular fibre

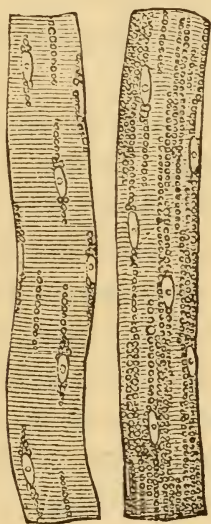


FIG. III.

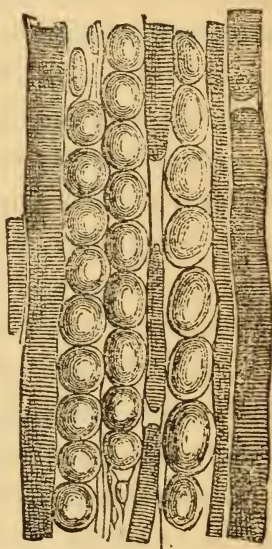


FIG. IV.

only by pressure upon it, while the former attacks the muscular fibre primarily. The cells of which the muscular fibrillæ are composed decay, and are gradually transformed into fat. It is by this process that the muscles pressed upon by bandages become atrophied after fractures. Perhaps somewhat after this manner also, the uterus resumes its normal dimensions after parturition. The two illustrations above from Rind-

fleisch serve well to explain the difference between the two conditions.

Fatty degeneration begins in the nuclei of the cells, destroys them first, and passes on until the whole cell is destroyed and converted into a mass of fat. The disease does not spread from a single centre, taking all as it goes, but many cells are attacked at once, these many centres of decay enlarging and gradually approaching each other until the whole tract affected becomes converted into fat.

Healthy muscular fibre displays under the microscope, transverse striæ, but when degeneration begins, these striæ become gradually obliterated, and the fibre by degrees comes to be marked longitudinally, these linear tracings representing oil molecules arranged in rows within the walls of the fibrillæ which compose the muscular fibre. Later still, this linear striation is itself obliterated, and nothing is seen but a homogeneous mass of fat, but death is apt to occur before this stage is reached.

As degeneration progresses, the affected part changes to a dirty yellow color, the deep blue coronary veins showing plainly in contrast with the surrounding yellow tissue, which has now become soft and easily torn.

Fatty degeneration and fatty infiltration, however, are very frequently found existing together. Fig. III is an example of this combination. The fatty deposit in the areolar tissue is distinctly shown, while the fibres themselves are diseased in some portion of their extent, as shown by the longitudinal markings, other



portions being sound, and the transverse striæ still perfect.

Fatty degeneration affects the ventricles in preference to the auricles, and the left ventricle in preference to the right. It may be evenly distributed over the affected portion, or may be found in patches of varying size, from that of a pin's head to that of a pea; or a single small portion may be affected, corresponding to the distribution of one of the branches of a coronary artery, which has become occluded by an embolus. It is frequently seen upon the outer surface of one of the ventricles, corresponding in depth to the extent of the inflammation in myo-pericarditis, and in such cases it may be merely superficial, or may effect almost the entire diameter of the cardiac wall. Usually the tract invaded exhibits the different stages of the affection at the same time, the centres of origin being most advanced.

*Conditions.*—Fatty degeneration of the heart is a disease rather of advanced life. Of the 95 cases of Hayden, and of the Dublin and London pathological societies, 24 were under 40 years of age, 26 from 40 to 50, 8 from 50 to 60, 28 from 60 to 70, 7 over 70, and 2, age not stated.

Of these 95 cases, 64 were males and 31 females, so that the number of males affected in proportion to the females would seem to be a little more than 2 to 1. Before the age of 50, the number of females affected predominates.

Occupation seems to have but little to do with it as



far as known. The common supposition that those leading sedentary habits would be more likely to be attacked by it, arises from the confusion which formerly, and even now, exists in the minds of many, as to the nature of the affection, fatty degeneration and fatty deposit having been formerly considered as identical.

*Etiology.*—The various causes are not known to a certainty, but the greatest is supposed to be the entire or partial cutting off of the blood supply from the part affected. The reason for this supposition is, that the two phenomena are so frequently found existing together, the defective circulation usually, if not always, manifesting itself first. This obstruction may be a consequence of an atheromatous condition of the arterial system in general, it may proceed from an embolus having lodged in one of the coronary arteries, or a branch of one of them, either entirely or partially cutting off the circulation in the part beyond it, or a portion of the pleura or pericardium may be drawn so tightly over a coronary branch as to prove an obstruction. Perhaps the most frequent cause of deficient blood supply is from an atheromatous aorta, which, having lost its elasticity, is no longer capable of recoiling with sufficient force to fill the coronary arteries. Degeneration may occur also in consequence of poverty of blood from deficient nourishment. Another cause is found in myocarditis. Indeed, speedy fatty degeneration constitutes one of the leading dangers in the latter affection.

Ordinarily, the rapidity with which the degeneration progresses, seems to be proportionate to the degree of obstruction. When that is almost complete, the degeneration is very rapid in its course. If an embolus lodges at the mouth of one of the coronary arteries in such a way as to cut off the circulation, death may occur at once without degeneration first taking place.

Other causes are certain poisons, among which are phosphorus and several of the mineral acids, also typhoid, typhus and exanthematic fevers.

*Physical signs.*—The great characteristic of fatty degeneration is absence of all physical signs.

Inspection shows nothing but the absence of action, and where there is any considerable degree of degeneration, the apex beat cannot be seen even in the thinnest persons.

Pressing the hand upon the precordium betrays the same loss of power. The impulse either cannot be felt at all, or if degeneration is consecutive to hypertrophy and dilatation, there is felt only the peculiar rolling motion of dilatation enfeebled by decay, but this motion belongs to the undegenerated fibre only, for a heart far advanced in fatty degeneration can not be felt, even with the greatest amount of enlargement. Its debility is so great that it is not capable of making any perceptible impression upon the chest walls, even in the prone position, while in those cases in which the apex beat is only concealed by effusion within the pericardium, it becomes again perceptible

## DISEASES OF THE WALLS OF THE HEART.

when the patient leans decidedly forward, or lies upon the face.

Percussion yields no sign that would betray anything abnormal, for the heart generally undergoes no change in its dimensions, except in cases of dilatation from previous hypertrophy, and this dilatation would likely have occurred in the absence of degeneration, as well. If we find the limits of dullness normal, we know that the loss of apex beat can not be accounted for by pericardial effusion, and thus, the only sign of value is a negative one.

The signs educed by auscultation are of more importance. Upon applying the stethoscope, we find the first sound either entirely absent or greatly diminished in intensity. The muscular element is generally wanting altogether, which leaves only a valvular element, and this more feeble than in the healthy heart. In dilatation without fatty degeneration it is different, for there we have the valvular element of the first sound sharp and ringing, loud, and widely transmitted over the chest. In dilatation with fatty degeneration of moderate degree, the first sound is louder and more widely diffused than in degeneration without dilatation, in consequence not only of the greater excitability of the heart, but of the thinness of the walls through which the sounds have to pass.

The second sound in fatty degeneration is still clear but feeble, though more distinct than the first.

From the physical signs above given, it is evident that the leading characteristic of fatty degeneration is



absence of action, an acknowledgment on the part of the heart, of its own debility. Palpitation does not belong here. It is a symptom of weakness with excitability, and therefore of dilatation, while inaction with its consequent vertigo and syncope, is a characteristic of degeneration. If we have palpitation in a case in which fatty degeneration is present, it is usually where the latter condition has set in consecutively to hypertrophy and dilatation, and the palpitation belongs not to the decayed fibres, but to those which are as yet undegenerated.

The irregularity of degeneration is not that of palpitation, but is of a more soft, undulating character, with prolonged halts. Neither is it so active. It is distinguished from that of nervous origin by being aggravated on exertion. This phenomenon is not frequently met with, the pulsations being usually regular.

Intermittency is also occasionally present, and this may be an intermittency of the pulse alone, the heart beating a feeble beat in the intermission, not strong enough to be heard by the stethoscope, or it may be an actual intermission of the heart's pulsations, and consequently of both the heart and pulse. When present, it is always increased by exertion.

As the fatty degeneration of old people is almost invariably preceded by atheroma, the temporal artery can in such cases usually be seen enlarged and tortuous.

The pulse may be irregular and intermitting, but is



much more frequently regular, and slower than natural. Except where aortic regurgitation is present, it is found to be very feeble, and, if the degeneration is very far advanced, may be altogether imperceptible. It may appear to be stronger than it really is, in consequence of an atheromatous condition of the radial artery.

The skin is usually pale and of lower temperature than the normal.

In the arcus senilis, the eye presents to us a symptom of some little diagnostic value. This is an opaque circle found at the junction of the sclerotic and cornea. There are two kinds; the one, according to Fothergill, of a bluish tint, and rather well defined, with an eye of bright and lively expression, indicative of a hearty old age; the other dull, yellowish and ill defined, associated with a dim eye and cloudy cornea, an unsteady, tottering gait, and a very feeble, perhaps irregular and intermittent pulse, and feeble heart contractions. This last is the arcus senilis indicative of fatty degeneration, and is itself composed mostly of fat globules. It is only occasionally met with, and is no more indicative of fatty degeneration of the heart than of any other organ, but is valuable as an indication that the cardiac lesion, if any be present, is fatty degeneration.

Hayden regards œdema of the lower extremities, extending up to the external genitals, as conclusive evidence of fatty degeneration of the heart and kidneys

combined, an opinion which to me seems to be erroneous.

In the beginning, the subjective symptoms peculiar to this lesion are generally wanting. Later, these may be present in a marked degree, as vertigo, syncope, pseudo-apoplexy, impaired mental action, muscular tremulousness, breathlessness, angina pectoris, rhythmical irregularity, etc.

All of these symptoms, with the exception of the last two, are the result of an enfeebled circulation, while the first five mentioned are due to cerebral anæmia. It is doubtful if any one ever died of fatty degeneration without cerebral anæmia manifesting itself in one manner or another.

First and most constant among the symptoms due to this condition is vertigo. This is present in a vast majority of cases, and very frequently amounts to complete syncope. It is always worse after every exertion, it being a very common thing in the later stages for the patient to faint completely away while running to catch a train, or suddenly rising up, or straining at stool. These patients most frequently die in this way, although the exertion is often unknown to any but the patient himself, who does not live to tell of it.

Another manifestation of this cerebral anæmia is pseudo-apoplexy, with sometimes actual convulsions. The attacks are short, and may be frequently repeated. The features are usually pale and tranquil, but may be livid from spasm of the muscles of respiration. The

patient is left without paralysis, a fact of great importance, as helping to distinguish this from genuine apoplexy.

Impaired mental action is another of the effects of cerebral anæmia. The heart being unable any longer to send a sufficient amount of blood to the brain, the latter organ becomes chronically anæmic, while if the pulse intermits, there is an acute exacerbation of this anæmia. In order that the brain may perform its functions properly, it must be supplied with its due share of blood. When any great task is to be performed, or any great mental effort is required, the heart is overpowered by the demand upon it, it intermits, and the subject of the degeneration falters, his mind is incompetent to the task, his judgment errs, and he perhaps acquires a reputation for fickleness.

Another effect is muscular tremulousness, and an unsteadiness of gait, as if the patient lacked confidence, with a certain timidity, an avoidance of all shocks, an inclination frequently to catch at the nearest object for support, as the heart falters in its task. It is a change in the physical, similar to that in the mental condition.

Rhythmical irregularity of respiration is a very common symptom of fatty degeneration, the respirations now gradually dying away so as to cease entirely for a quarter of a minute, now gradually increasing to full inspirations, again to fade away as before, the duration of the entire cycle being about one minute, during which time there may be counted about twenty-



five or thirty respirations. The pulse meanwhile continues at its usual rate, without regard to the changes in respiration. This is sometimes called the "symptom of Cheyne," from the name of him who first observed and recorded the phenomenon.

As to the origin of the last mentioned symptom, several different explanations have been given by different authors, perhaps the most rational one being that of cerebral anæmia.

The breathlessness of fatty degeneration is different from cardiac asthma, often met with in dilatation, right side hypertrophy, valvular disease, etc., being shallow, hurried, *without effort*, with anxious, wild look, while the other is laborious, and often accompanied by whistling and wheezing.

Angina pectoris is present in perhaps one tenth of the cases, and is of no diagnostic value unless it be accompanied by the other symptoms of degeneration, as the feeble cardiac action and feeble pulse, and even then is not at all conclusive. In this distressing affection the patient is attacked suddenly with great oppression and weight upon the chest, a pinched feeling, and with very shallow and quick respiration, but without effort or wheezing in the *bronchi*; the face has upon it a look of the greatest terror, is pale, and great drops of sweat roll down the forehead and temples. Pain may or may not be present. The patient and friends are very much alarmed, and expect death to occur at every moment, and indeed the first attack may prove fatal, or he may live to have several of



these attacks, and afterwards die in some other manner.

This is to be distinguished from cardiac asthma by the fact that in the latter the lips are blue from imperfect oxygenation of the blood, and the muscles of the chest are in violent action, struggling for breath, while in angina the lips are pale, and the breathing not labored but very short and rapid, the chest walls remaining more quiet than natural.

The history of a case of fatty degeneration is usually unmarked in its early stages, when it is difficult or impossible to diagnose. The patient himself does not know of its existence for a long time, or keeps the matter a profound secret. Usually the first knowledge that he or his friends have of it, is after some sudden surprise, some accident, or some violent exertion, when the cardiac debility manifests itself through threatened syncope, or possibly inordinate breathlessness.

The patient is generally beyond the middle age, with gray hair, or entirely bald perhaps. As the disease progresses, he becomes incapable of violent exertion, and studiously avoids all great efforts, both mental and physical; his eye is dull, his gait unsteady and faltering, with much manifest weakness, as he stops to rest against the nearest object. There may be cough, without much expectoration, and a feeble and petulant voice. The patient is child-like, mentally, physically, and morally. He is like a man who is prematurely decayed. His condition has been described as one of ripeness merging into rottenness.

The urine is scanty, of high color, and with tube casts and more or less fat globules, while the heart sounds are deficient in intensity, so that the first sound in the later stages can scarcely be heard, if at all, and the impulse is usually altogether imperceptible. At this time, also, we have œdema of the lower extremities, gradually extending upward towards the body. It must not, however, be supposed that every case of fatty degeneration of the heart possesses all these peculiarities, for many of them may be absent.

The less active a life the patient leads, the longer will he be likely to live, and the longer he lives the more of these symptoms will be developed, and the more must he suffer before death comes to his relief. Women, more quiet than men, and less liable to exert themselves violently or suddenly, usually live longer with this disease, and, accordingly, suffer longer, and in the later stages more intensely. The most frequent cause of death is from sudden motion, as turning over in bed, rising up, or a sudden start, as from painful or joyful news, or straining at stool. In this case the heart may become paralyzed, or a rupture may occur in its walls. Indeed, this latter termination is not by any means so rare as might at first be supposed. It is much better for the patient that he should die thus suddenly than to suffer on, for many live until all the signs of venous congestion in the encephalon have manifested themselves, as drowsiness, coma, and hideous, horrid dreams; and until the bronchial tubes so

participate in the general venous congestion as to be completely closed with mucus.

*Prognosis.*—The prognosis as to time may vary greatly; in some cases the patient may live for many years, while others may prove speedily fatal. Of ninety-five cases tabulated by Hayden, the shortest duration of illness was four days, and the longest forty years. The patient may not die of fatty degeneration at all, but of some other disease, which has been rendered more dangerous by the cardiac affection.

*Treatment.*—Most writers on the subject speak of this affection as absolutely incurable, but I believe that Homœopathic physicians are not accustomed to regard it as such; and if a muscle can become atrophied by fatty degeneration, the result of the pressure of bandages, and afterward recover its original size and strength, why may not fatty degeneration in the heart walls, when the result of a curable anæmia, or of typhoid or exanthematic fevers, be as truly curable? And when this is not possible, it seems to me that we should, in the early stages of many cases, be able to at least stay the progress of the malady. We are certainly not without remedies which possess a similarity to it in their pathogenesis. One important reason for our lack of success in the treatment of this disease, is the fact that the physician is not usually called upon to treat it until it is far advanced.

The object of treatment is 1. To cure, or at least arrest, the progress of the degeneration. 2. To strengthen that portion of the heart walls which,



though weakened, are structurally sound, and thus to remove the consequences of a defective circulation.

Among the remedies most likely to be found useful are the following:

*Arsenicum* has, among other symptoms, violent palpitation of the heart, with quick, weak, and irregular pulse, tremulous weakness, and irritable heart's action. Also, very feeble action of the heart; small, slow pulse; pale skin; flabby muscles, especially in stout persons; difficult breathing, especially when going up stairs or up hill. Suppressed herpetic eruptions; after scarlatina; dropsical tendency.

Upon theoretical grounds, this remedy has often been recommended for fatty degeneration of the heart, but the expectations of its advocates have not been fully realized, and there is sufficient reason for this disappointment, for those symptoms in its pathogenesis which seem most to point to its use in this disease, are merely the result of its effect upon the ganglionic nervous system. It is, however, frequently indicated in those affections which often lead to or accompany fatty degeneration; notably, in valvular disease, dilatation, and angina pectoris, and here it has proved to be of no little value.

*Aurum*.—Frequent attacks of anguish about the heart, with tremulous fearfulness; very violent beating of the heart after exertion, with great agony; when riding or walking, palpitation compels to stop; palpitation with irregular, intermittent pulse, and strong breathing, or suffocative oppression of the chest.



Loathing of life; melancholy, despondency, suicidal tendency; great nervous weakness, with utter despair. Scrofulous or syphilitic subjects; after mercury.

The cardiac symptoms in the pathogenesis of Aurum, so far as known, are not those of fatty degeneration, but of such cardiac debility as might proceed from dilatation or inflammation, for a heart far advanced in fatty degeneration does not palpitate, nor is it capable of any violent action. No one would care to push the proving upon the human subject to such an extreme as to produce fatty degeneration, yet it is not at all improbable that it has the power to produce such change. Something more should yet be learned in regard to this remedy by provings upon the brute creation.

*Calcarea carb.*—Vertigo on ascending a height; much dandruff on the scalp; dull headache in the morning, head cold; cannot sleep after 3 A. M., acidity of the stomach, which is bloated; chronic diarrhœa with chalk-like stools. Leuco-phlegmatic temperament, scrofulous constitution, inclined to grow very fat; flabby muscles; cold hands and feet; feet feel as if she had on cold damp stockings.

*China.*—This remedy has no such cardiac symptoms as would indicate it in fatty degeneration, yet it may be of use in some cases in which the degeneration is the result of general anæmia, when it can be traced to a malarial origin, or after loss of animal fluids.

All the symptoms aggravated by the slightest touch; abdomen feels full and stuffed, eructations give no re-

lief; diarrhœa of undigested food, mostly at night, infarctions of the liver, with jaundice.

*Cuprum*.—Baehr gives the following as among the symptoms produced by Cuprum in cases of poisoning, "Pulse irregular, small, easily compressible, and intermitting, accompanied by excessive muscular debility; the beats of the heart are scarcely or not at all perceptible; sounds of the heart are indistinct; dyspnœa; feeling of anxiety, disposition to faint."

These symptoms are certainly very different from those found in our works on materia medica, which can scarcely be said to point to the affection under consideration at all. I have had no experience with it.

*Digitalis*.—Very feeble, irregular action of the heart, with feeble, small, intermitting pulse; great prostration, fainting on the least movement, even lifting the arms, with palpitations, coldness of hands and body, and dreadful weak feeling in pit of stomach, with or without convulsions or syncope; profuse expectoration of bloody mucus, and vomiting of food; sensation as if the heart would stop beating if she moved, with fear of impending death; effusion into the pericardium; urine scanty, with brick-dust sediment.

It is not probable that this remedy has any power either to produce or cure fatty degeneration in a direct manner; yet, by its power to strengthen that portion of the heart muscle which, though enfeebled, is structurally sound, it is capable of affording great relief in those cases which are complicated by dilatation, and, when dilatation has been the cause of such

structural change, may even stay the progress of the degeneration; the enfeebled heart walls being strengthened by it, and thus rendered capable of more vigorous contractions, which will be followed by a more vigorous recoil, better filling of the coronary vessels, and consequently more perfect nourishment of the diseased organ.

*Ferrum*.—When structural change results from anæmia or chlorosis, this remedy may be of use; only, however, as an intercurrent, as it is not Homœopathic to the cardiac degeneration itself.

*Iodium*.—Vertigo, immediately after rising from a seat or bed, or on sitting or lying down after slight exercise; palpitation increased by movement; fainting spells; fluttering sensation in the heart; constant, heavy, pressive pain in cardiac region; tendency to melancholy. Skin rough and dry; dirty yellow; disposition to glandular enlargements.

*Phosphorus*.—Disease of right heart, with consequent venous stagnation; dyspnœa, tightness across the chest; great weakness with inability to exert himself; very great sensitiveness to external impressions; hæmic murmur at base of heart; anæmia; disposition to pulmonary affections; congestion of lungs; tight cough; painless diarrhœa.

This remedy corresponds pathologically to fatty degeneration of the heart, and when indicated by the symptoms as well as the pathological condition, should be capable of doing much good. Reliable clinical experience with it, however, as with other remedies, is



not as abundant as we could wish, partly, perhaps, in consequence of the difficulty of diagnosis in the early stages, and the yet far too frequent confounding of fatty degeneration with fatty deposit. Dr. Lade reports the case of a woman aged fifty-four, who was at least very greatly benefitted, if not cured, in eight months time, by Phosphorus and a generous diet. The arcus senilis entirely disappeared.

*Phosphoric acid*.—Palpitation, always after starting up in sleep. May be useful in incipient degeneration, with very excessive perspirations especially at night; painless diarrhoea; urine contains heavy deposits of earthy phosphates. After onanism or excessive venery.

*Phytolacca*.—Shocks of pain in cardiac region, pain goes into right arm; weakness with heaviness near heart, worse during expiration, cannot get to sleep again; heart's action weak, with constipation; great exhaustion; pulse small, weak, and irregular.

*Plumbum*.—Prominence of precordium; hypertrophy with atheroma of aorta, followed by dilatation, especially when connected with chronic Bright's disease; basic murmurs, especially systolic, and either obstructive or hæmic. Obstinate constipation; difficult micturition; urine dark colored and scanty, evacuated by drops; urine albuminous, dark, flocculent, containing blood corpuscles.

Baehr gives the following group of symptoms produced by lead poisoning. "The impulse of the heart is very feeble, even imperceptible, intermittent; sounds of the heart indistinct; palpitations attended with



excessive dyspnœa; pulse very soft, easily compressible, intermitting; weak, fifty to sixty to the minute, less frequently over a hundred, after which it is scarcely perceptible; heart flabby; sudden paralysis of the heart; fainting fits during every exertion; also attended with slight convulsions; extreme muscular debility, and oppression from the least motion; despondency and dread of death; œdema of the skin."

In all cases it is necessary to keep the patients digestive apparatus in as perfect a condition as possible; not by the use of stimulants, but, when anything is required, by a properly chosen Homœopathic remedy. If he has any other disease which is debilitating him, let that not be merely suppressed, but if at all practicable, cured as speedily as possible. He should have a nourishing diet, easily digestible, but not too much of it, for the excess would not only do no good, but much harm by impairing digestion. Keep him as quiet as the demands of his debilitated heart would seem to require. All sudden surprises and shocks, all violent exertion, running and excitement, should be avoided.

*Fatty Infiltration.*—As already shown, this is an entirely different affection from fatty degeneration, and spends its force on the connective tissue and not upon the muscular fibrilæ. It is an infiltration of fat globules in the connective tissue, and is injurious in consequence of the pressure it makes upon the muscular fibres between which it is deposited, producing atrophy, and frequently also fatty degeneration there-

in. As portions of these fibres become destroyed, the heart muscle of course becomes weakened.

The fatty deposit may be scattered through the substance of the heart, or it may begin upon the surface, and follow the connective tissue inwards, destroying by pressure all before it.

*Diagnosis.*—This is always difficult. The patient is generally quite fleshy, while in fatty degeneration he is more apt to be spare. With infiltration and the fatty deposit upon the surface, there is usually considerable increase in the dimensions of the heart, but this is not generally perceptible in consequence of the excessive adipose upon the surface of the body.

The physical signs of the heart and pulse are usually the same in this condition as in pure fatty degeneration, and to distinguish between these two affections to a certainty is absolutely impossible. The best that can be done is to form a relative opinion from the history of the case and the general condition of the patient. Both are often found existing together.

With fatty deposit, cardiac asthma generally takes the place of the angina pectoris often found in fatty degeneration, although I remember treating a case of fatty heart not long since, in which there was genuine angina, which the proper remedies relieved for a year at least, and I have no knowledge that it ever returned. There may, however, have been considerable degeneration in this case in addition to the infiltration. The general health in the mean time was greatly improved.

The *prognosis* of pure fatty deposit is somewhat better than that of fatty degeneration.

*Treatment.*—This should be principally hygienic, its object being not merely to prevent decay, for usually there is none except what is secondary to the infiltration, but to reduce as far as possible, the tendency to obesity. To this end:

1. The patient should be deprived, to a great extent, of such articles of food as contain a large proportion of fat, of sugar, or of starch, and should substitute for them a nitrogenous diet, consisting largely of coarse and not over nutritious food. Let the amount of bread be limited; and what the patient does eat should be made from a good article of unbolted flour, as containing less starch than that made from fine flour. Fruits, and vegetables except potatoes, should form a large portion of his living. Lean meats are not particularly injurious. He should always leave the table before the appetite is fully satisfied.

2. Fully as important as the matter of food is it, to remember, that the less fluid the patient takes into, his stomach, the better, whether at meal time or between meals, for water is an important element in the formation of fat.

3. As soon as he is strong enough, let him take moderate but *never violent* exercise, and this every day, as much as he can endure without too great fatigue.

*Arnica* has been recommended, but I would sooner think it of use in degeneration.



If fatty degeneration is suspected, let the treatment of that affection be combined with the hygiene of this in such a way as the physician's best judgment may dictate.

*Granular softening* has been fully demonstrated by Hayden to be merely the first stage of fatty softening or degeneration.

*Simple Softening.*—This is a condition entirely distinct from fatty degeneration. The tissue is somewhat darker than natural, is soft and easily torn, flabby, and according to Louis, capable of being moulded into various forms like a wet cloth. That there is no change of structure is evident from the fact that when not terminating fatally, it is only of temporary duration.

It is found in typhoid and typhus fevers. It generally appears about the sixth day, and continues thereafter from seven to nine days.

The signs are those of cardiac debility. The impulse is generally very feeble or altogether wanting, and the first sound almost or entirely inaudible. The pulse is generally quite rapid, from 140 to 150 to the minute, and feeble but regular. These symptoms do not manifest themselves all at once, but increase in prominence as the softening progresses, and with a returning healthy condition of the heart walls, again gradually diminish. The pulse may now even fall far below the normal standard to which it afterward rises.

Vertigo is common, with disposition to faint, even to complete syncope on rising up.



Simple softening is also occasionally met with in delirium tremens, and when its signs are observed, they should be regarded as exceedingly unfavorable, and perfect quiet be maintained as far as possible, lest sudden death may ensue from overpowering the heart.

For the treatment, I would suggest Ars., Bry., Digit., Fer. and Phos., the indications of which, with the exception of the last mentioned, will be found in the treatment of hypertrophy and dilatation. Phosphorus is especially indicated in slender persons, if there is a disposition to bronchial or pulmonary affections, not produced by heart failures, sensitive to impressions, painless diarrhœa. If fatty degeneration accompanies or follows, study the remedies under that head.

## CHAPTER VII.

### CANCER AND WOUNDS OF THE HEART.

The heart is very seldom primarily affected with cancer, but has been frequently known to participate when the disease has its original seat in some other organ, as the lungs or mediastinum. The medullary variety is of most frequent occurrence.

The symptoms vary according to the distribution of the growth. Sometimes it produces murmurs by constricting orifices, or drawing valves out of shape so as to permit regurgitation; creating hypertrophy or dilatation of moderate degree, or by displacing the heart entirely, or the apex only. It is liable to be confounded with aortic aneurism, but in the latter there will be found a perceptible impulse over the tumor. In cancer the patient usually presents a general cachectic appearance. There is likely also to be present, cough and more or less hæmoptysis.

It is perhaps unnecessary to state that the affection, when found, will be incurable, and even palliation to any considerable extent, will be found quite difficult if not impossible.

*Syphilitic Nodules.*—These are from the size of a small pea to that of a cherry, yellow, solid in structure, and situated in the cardiac walls. According to Oppolzer, they are liable to soften and break into the

cavities of the heart. They may be most properly classed as tertiary symptoms, and are very rarely met with. Atrophy is occasionally found as a complication. A diagnosis is impossible.

*Hydatids* have been met with in the walls of the heart, but are extremely rare. There are no symptoms which are pathognomonic of their presence.

*Tubercles* have also been found both in the pericardium and in the walls of the heart, but it is doubtful if they are ever present except as a complication of pulmonary tuberculosis.

The treatment should be similar to that for tuberculosis elsewhere, preserving the vitality, improving the digestion, but always observing the law of similars in choosing the remedy.

*Aneurism*.—This is a rare lesion. It is most frequently seen in the left ventricle, and oftenest at the apex. It occurs in males more frequently than in females, the proportion being, according to Quain, as three of the former to one of the latter. Others make the difference much greater, but this is perhaps very near the truth. While childhood is generally exempt, it would not seem to occur by preference at any particular period of adult life.

The term cardiac aneurism was formerly applied to the condition now known as dilatation, but as now used it means such a yielding of a portion of the wall of one of the chambers of the heart, as to produce a sac communicating with that chamber. This sac generally projects from the cardiac surface, and may attain

to a considerable size, sometimes becoming larger than the chamber with which it communicates.

Among the causes of cardiac aneurism are ulcerative endocarditis, chronic endo-myocarditis, affecting only a limited portion of the walls, partial rupture of the wall from within outward, and bursting of an abscess into one of the chambers.

In the majority of cases, the sac walls would seem to be composed of all the structures of the heart, while in many the endocardium, or the muscular structure, or both, may be lacking, leaving only the pericardium.

The *diagnosis* during life is very difficult, if not impossible. The symptoms are those of heart failure generally, as dyspnœa, anxiety, precordial pains, palpitation on exertion, dropsy, and threatened or actual syncope. Besides, we are apt to have increased dullness and possibly fluctuation over the sac. Schroetter thinks the existence of any characteristic murmur highly improbable.

This affection cannot be said to be absolutely incurable in all cases, yet the prospect is not by any means encouraging. In those rare instances in which recovery takes place, it is by solidification of the contents of the sac. The most common termination is in rupture of the sac, although in some instances the patient may live for many years.

The treatment is chiefly hygienic. All excitement, both mental and physical, should be avoided, so as to promote the deposit of the fibrine of the blood in layers on the inner surface. All spirituous liquors



should be absolutely prohibited, and a nutritious diet prescribed, while perfect digestion is secured by the administration, when necessary, of the proper Homœopathic remedies.

*Wounds of the heart.*—The right ventricle being situated more anteriorly, is more likely to be wounded than any other portion of the organ, this part being wounded in something more than one-third of the cases. The wound is generally made by some foreign body, as a bullet, knife, saber or bayonet, penetrating from without, while wounds produced by some object passing down the œsophagus, as a fish-bone, pins or needles, or similar objects, are likely to implicate the left ventricle.

The heart muscle is sometimes wounded without the pericardium being ruptured, that membrane being merely carried forward by the foreign body into the wound.

Although in general, immediately fatal from hæmorrhage, such a result is in some cases postponed for some time, or until reaction takes place, the dislodgement of a clot from the wound favoring hæmorrhage and speedy death. In other cases, the pressure of the blood effused into the pericardium produces paralysis of the heart, and consequent death. Later still, if the patient survives, death may ensue from traumatic inflammation of the heart and pericardium.

A few cases have been known in which recovery took place. Brugnoli made a post-mortem of a man who lived nearly twenty years after having a saber

thrust almost through his heart, passing through the right ventricle and the interventricular septum, leaving a quite distinct mark on the mitral valve. Bullets have become encysted in the substance of the heart, and remained there for many years, death finally taking place from some other cause. The more oblique the wound, the more favorable the prognosis. Wounds of the auricle are perhaps always fatal.

The *diagnosis* is not always easy, as all probing must be avoided, lest clots in the wound should be broken up, and the hæmorrhage renewed. In case, however, the pericardium should be so filled with blood as to paralyze the heart's action by its pressure, the coagulum may be carefully removed.

Ice, in the beginning of all cases, should be freely applied in order to favor coagulation and suppress hæmorrhage. To this treatment may be added Arnica, both locally and internally, and if inflammation sets in, as it is likely to do where the patient lives long enough, Aconite also should be given. Stimulants should be absolutely prohibited, as increasing the heart's action, and consequently favoring both hæmorrhage and inflammation.

## CHAPTER VIII.

### ENDOCARDITIS.

As its name indicates, the seat of the inflammation in endocarditis, is the endocardium, which is continuous with and corresponds to the lining membrane of the arteries.

We shall first consider acute endocarditis, and include under this head, the acute form, both simple and ulcerative, and its immediate consequences.

It does not usually occur as an idiopathic affection, but in the course of some other disease, as rheumatism, Bright's disease of the kidneys, eruptive and typhoid fevers, etc. It will then be seen that the disease which it usually accompanies are those in which there is some kind of poison in the blood, but just how these poisons act upon the endocardium, or whether they are in most cases at all the cause of the inflammation or not, is not known to a certainty. It is not unnatural to suppose, however, that they should have a more or less deleterious effect on this structure when coming in contact with it. This supposition receives support from the fact that in pyæmia, where the blood has an acid reaction, are found relatively the most cases of this disease.

As to its etiology, perhaps rheumatism is in some way responsible in by far the larger portion of the

cases. It is supposed by different authors, to be present in that affection in a proportion varying from 10 to 80 per cent. Judging from my own experience, I should suppose the former to be much nearer the truth. Very much depends upon the treatment of the rheumatism. If this is good, the disease is likely to be thrown off before the endocardium has had time to become affected; although, in rare cases, the endocarditis may be the first and indeed the only symptom of the rheumatic affection. Perhaps next to this as a cause, is Bright's disease, and after that, the eruptive fevers, typhoid and typhus, puerperal fever, phlebitis and pyæmia.

As to the relative frequency of endocarditis and pericarditis, various estimates have been made. One cause of this difference of opinion is that some consider the existence of valvular disease a positive evidence of pre-existing inflammation of the endocardium, which it certainly is not. Keeping this fact in view, pericarditis will be found to be of the most frequent occurrence, while the combination of the two affections is more frequently met with than either one existing alone.

Endocarditis occurring during foetal life is generally found upon the right side, while after the child is born and respiration established, it is generally confined to the left side. The chief exceptions to this are those cases which result from phlebitis and pyæmia, when the right side is the part generally affected.

The mitral valve with its immediate neighborhood



is the most frequent seat of endocardial inflammation, being implicated in perhaps five sixths of the cases, and next to this the aortic valves, in many cases both being implicated.

The first stage, or stage of injection redness, characterized by branching vascular points, must not be confounded with that redness frequently met with after death when the heart has been healthy, and which is the result of stain from the coloring matter of the blood. The latter discoloration is smooth, more uniform, and frequently of a deep hue. In endocarditis the membrane is swollen considerably, especially upon the free edges of the valves, and studded with points of granulation. This granulation is liable to be confounded with the aggregations of fibrine upon the surface of the membrane, mostly about the valves, the result of the adhesion of the fibrine of the blood as that fluid rushes over it, and which are frequently met with in endocarditis, the granulations and fibrinous deposits often being intimately connected. These fibrinous deposits are occasionally washed loose and carried on by the blood current until the arteries become too narrow for their further passage, when they form an obstruction to the circulation in the parts beyond. When this takes place it is called embolism, and when one of the cerebral arteries is the seat of the obstruction, paralysis is likely to result. The embolus may be dissolved and the circulation restored, when the paralysis ceases. The villous excrescences which are the result of the inflammation

are liable to undergo granular degeneration and disintegration, when they may also be washed away, leaving superficial ulcers which may end in perforation or in valvular aneurism.

The inflammation is not generally confined to the endocardium alone, but extends to the substance of the heart, producing more or less myocarditis. When this extends to any considerable depth, it may result in aneurism by weakening the walls at the point affected, or there may be a simple weakening of the wall at a given point, not sufficiently circumscribed to amount to aneurism, but causing it to give way only at the acme of systole. If this takes place at the origin of a papillary muscle, it will prevent the perfect closing of the mitral valve, and give rise to mitral regurgitation, with its attendant systolic murmur. In other instances the papillary muscles are themselves so involved as to produce shortening and irregular action in them. This also prevents the mitral valve from closing properly, and with a like result. It will thus be seen that we may have a systolic apex murmur from regurgitation, without distortion of the mitral valve from disease within itself. I cannot conceive, however, of a similar endocardial murmur from simple roughness of the endocardium and without any mitral regurgitation.

In still other instances the valves themselves become distorted by the inflammatory process. They may be simply roughened at their free edges, so as not to close perfectly, yet not admitting of any considerable de-

gree of regurgitation, the first sound merely being somewhat prolonged, or the free edges of the two segments may be agglutinated together from the circumference towards the centre of the opening, thus partially closing it, making an obstruction to the blood stream coming in from the left auricle, and giving rise to a murmur pre-systolic in time, or they may be contracted so that their free borders are drawn apart, preventing the complete closure of the orifice, or the tendinous cords may be glued together and contracted so as to produce a like result. In either case, mitral regurgitation with its attendant murmur will be present. This adhesion of the two valve segments and their retraction into the ventricle, are frequently both found to be present in the same case, and we thus have what is called a funnel-shaped mitral, giving rise to obstruction and regurgitation at the same time, and causing a double murmur, the first, pre-systolic, immediately followed by another, systolic, but both heard loudest at the apex.

Acute endocarditis often runs its course without giving any sign to the patient himself, as the rheumatic pain in other parts absorbs his whole attention. Besides, the endocardium being poorly supplied with nerves, but little pain is felt. The same holds true in pericarditis. On this account there is no tenderness on pressure unless the inflammation has extended to the heart muscle, in which case there is generally also palpitation, with other evidence of cardiac debility, as a more or less excited action, and a weak and irregu-



lar pulse, with unwillingness to lie upon the left side. Should the myocarditis be extensive, there may be much dyspnœa, with pulmonary œdema, congestion and enlargement of the liver and kidneys, and general dropsy. The portion of heart muscle so affected, if examined early, will be found of a dead-leaf tint. Later, it exhibits the characteristics of fatty degeneration.

*Physical Signs.*—The heart sounds are usually interfered with to some extent in endocarditis, the most frequent alteration being a *systolic murmur near the apex*, from mitral regurgitation. This murmur, according to Hayden, is all but universally present in this inflammation. It may also be heard in many cases in which such inflammation does not exist. Hence, while it is not in itself a pathognomonic symptom of endocarditis, its absence is pretty good evidence of the absence of that condition. We also frequently have an aortic murmur, either systolic or diastolic, and sometimes a pre-systolic apex murmur from mitral obstruction.

Acute endocarditis is not usually found as a complication of acute rheumatism until after the fifth day. It is best, when beginning the treatment of a case of the latter affection, to examine the heart by auscultation and ascertain if there be a murmur present, and should there be such a murmur at the beginning of the disease, we may conclude that it is of long standing. In this case, the murmur being systolic apex, we shall be likely to find the second sound in the pul-



monary artery accentuated, with the right ventricle considerably enlarged, and probably also the left. Should there be no murmur at the beginning, and then after a little time a mitral murmur set in, the probability is that we have a case of acute endocarditis.

A mitral regurgitant murmur beginning in the course of acute rheumatism, and replacing only the valvular element of the first sound, while the muscular element is still normal, is indicative of simple endocarditis, while such a murmur replacing both elements of the first sound, would point to endo-myocarditis, the muscular element being either weakened or entirely lost in consequence of the debility of the heart walls occasioned by the inflammation.

Now and then, a case occurs in which the endocardial murmur is masked, as by extreme debility of the ventricle, or by extensive pericardial effusion, but those cases in which it may not be heard during some period of the disease, are certainly rare.

The distinction between an endocardial murmur and a pericardial friction sound has been already considered in the table of comparison in the chapter on pericarditis.

An endocardial murmur heard loudest at the base, although sometimes met with, is not generally indicative of acute endocarditis.

As the inflammation becomes more advanced, the pulse becomes weak and irregular in volume, and the lips blue; and with these signs are more or less un-

easiness, dyspnœa, feverishness and delirium. As it merges into the chronic form, the right ventricle becomes enlarged, and the second sound in the pulmonary artery is increased in intensity.

To fix upon any certain temperature as characteristic would be extremely difficult, as this depends largely on the various diseases upon which the cardiac lesion is attendant. Neither must one always expect to find its symptoms existing alone, for perhaps in a majority of cases, they are complicated with those of pericarditis.

When endocarditis occurs as a complication of the severer forms of disease, as pyæmia and the worst forms of septicæmia, it is likely to take on the *ulcerative* form. In such cases it may begin similarly to the simple form, but soon the epithelium is softened and washed away by the blood stream, leaving a smooth felt-like surface. On this surface, white, jelly-like mucous patches shortly appear which afterward decay and are washed away by the blood, leaving ulcers in their place. In other cases, the inflammation seems to be more deeply seated, and small yellow pustules appear, which afterward break and form ulcers.

Ulcerative endocarditis usually begins with one or more chills, succeeded by heat, perspiration, etc. Afterward the fever becomes more continuous and intense, the pulse ranging from 80 or 90 to 140 or 150, but is in every case small and irregular. The temperature is usually very high, from  $105^{\circ}$  to  $107^{\circ}$ , with delirium, drowsiness and stupor. The appetite is lost from the

beginning. Later, we may have vomiting and diarrhœa, with cramps in the calves before death. The liver and spleen are enlarged, with tenderness in both hypochondria, and the urine almost entirely suppressed. Eruptions and ecchymoses are frequently present. In short, the patient exhibits in a marked degree the symptoms of a severe typhoid, with the signs of great venous congestion from an obstructed circulation. If the inflammation is located upon the left side, auscultation will reveal the same sounds that are heard in simple endocarditis; if, however, the right side is the part affected, the sounds will of course be modified accordingly.

The one disease with which there is danger of confounding it is typhoid fever, and from this it is distinguished in the earlier stages by the repeated and prolonged rigors, and afterwards by the heart symptoms which soon manifest themselves.

The *prognosis* of *simple* endocarditis, as far as the immediate result is concerned, is good, in as much as it very rarely terminates fatally. It usually becomes dangerous only in a degree corresponding to the extent to which the heart muscle participates in the inflammation. Nevertheless, it is a serious affection, because of its disposition to become chronic, and of the resulting distortion of the valves and their appendages. Hayden says, that in consequence of the continued strain upon these in carrying on the circulation, a complete cure is never possible, but this statement should perhaps be somewhat modified.



The ulcerative form has an exceedingly unfavorable prognosis. It runs a very rapid course, and the patient soon dies in coma.

*Chronic Endocarditis.*—After an attack of simple acute endocarditis, the inflammation may pass away entirely, merely leaving the valves more or less distorted and incompetent, with their free edges somewhat thickened and the endocardium less transparent than natural, but the usual tendency is to pass into the chronic form, with slow and continual increase of connective tissue, and ever increasing contraction and distortion of valves. This is one of the two distinct forms of chronic endocarditis, and if the acute attack which preceded it has been an accompaniment of acute rheumatism, its seat is generally at the left auriculo-ventricular orifice.

The other form is more strictly valvulitis, and is the result of strain, usually from violent closure of the valves. Its most frequent seat is at the aortic orifice, and next, at the mitral. A good example of this form of chronic endocarditis is sometimes seen in the left auricle, where it is caused by the strain attendant on forcing the blood stream through a constricted mitral orifice.

In both forms, the new tissue which is the result of cell proliferation in the inflamed part, is subject to a species of granular degeneration or petrefaction, rendering the diseased structures rigid, brittle and rough, and sometimes giving a loud, rasping character to the murmurs heard.



When the mitral valve is the seat of the chronic inflammation, these two forms are generally found existing together.

Chronic valvulitis will be further considered under the head of valvular disease.

*Treatment.*—The treatment of endocarditis is not as satisfactory as could be desired; not that there is in most cases any special danger of an immediately fatal result, but because of the difficulty or utter impossibility of making the cure complete, and removing the products of the inflammation, and because also of the unsatisfactory character of our provings of medicines. Still, a great deal can be done by a carefully chosen remedy, especially in the simple acute rheumatic form, while considerable assistance may be given in many cases of chronic endocarditis, especially where it is a continuation of an acute attack. Even in the ulcerative form, we should not always despair of success, particularly if the case can be diagnosed early.

Our endeavor should be to make the acute inflammation as light and of as short duration as possible, and to prevent, as far as is in our power, all thickening or distortion of the valves. Whatever is beneficial in rheumatism, *may* be of use in the treatment of rheumatic endocarditis, but among these medicines are some which have an especial affinity for the heart, and these should be considered of the most importance. The remedy should be chosen not with reference to the heart alone, but the accompanying disease and conditions should be taken into consideration.

*Aconite*, in acute rheumatism more frequently Homœopathically indicated, and hence more efficient than any other remedy in that distressing affection, will be found very useful in the first few days, in those cases which are of rheumatic origin, especially if there be considerable thirst, dry, hot skin, and full and rapid pulse. Prepare a few drops of the first decimal dilution in half a glass of water, and give a teaspoonful at a dose every hour or two until the patient is better, or the symptoms have changed so that some other remedy is indicated.

*Arsenicum* may be of value in the ulcerative form, or when the inflammation is dependent on scarlatina or Bright's disease.

Violent palpitation of the heart with small and irregular pulse; irregular palpitation of the heart, but so violent that he imagines he hears it, accompanied with anguish; palpitation and tremulous weakness after stool; quick, weak and irregular pulse, sometimes wholly impercēptible; great debility; dropsical tendency; pericarditis with extensive serous effusion.

*Aurum*.—Frequent attacks of anguish about the heart with tremulous fearfulness; violent beating of the heart after exertion, with great agony; when riding or walking, palpitation compels to stop; frequent deep breathing, suffocative fits, with spasmodic constriction of the chest, face bluish red; drawing, cutting pain at the heart; rheumatic endocarditis; melancholy, suicidal mania; profuse perspirations.

Burnett, in his monograph on "Gold," relates sev-

eral cases of rheumatic origin, cured with this remedy.

*Baptisia* is important in the early stages, when the disease takes on a typhoid form. It is none the less applicable if ulceration be present. The skin is hot and dry, the pulse rapid and feeble; afterwards slow and feeble, the tongue thickly coated with brown fur, the mouth clammy, and there may be considerable thirst with nightly delirium and restlessness. Marked debility is present from the beginning.

*Bryonia*.—If there is a persistent friction sound from an accompanying pericarditis, it is very important that *Bryonia* should not be forgotten. Even where there is no pericardial inflammation, this remedy is very often required, and particularly when the patient is worse on rising up, and from the least motion, with great thirst for large quantities of water. Other indications are oppression at the chest with sense of suffocation, and a pulse weak and irregular in volume from accompanying myocarditis. It is most useful in the acute and sub-acute stages.

*Cactus grand.* is one of our best remedies in this disease. It is useful in all stages, but more especially in the sub-acute and chronic. The tincture and first dilution seem to meet with most favor, yet I have found benefit from the thirtieth as well. Among its symptoms are: Periodical attacks of suffocation, with fainting, cold perspiration on the face, and loss of pulse; palpitation of the heart continues day and night, worse when walking and at night when lying on the left side; dull, heavy pain in the region of the



heart, increased by external pressure; sensation of constriction in the heart, as if an iron band prevented its normal movement; great sadness, melancholy, irresistible inclination to weep.

*Digitalis*, so important in pericarditis, is of little use in simple inflammation of the endocardium. It may, however, be required if the symptoms indicate extensive myocardial complication, or if there is also pericarditis with considerable serous effusion. Indications are, very feeble, irregular action of the heart, with small, feeble and intermittent pulse; great prostration, fainting on the least exertion or movement, even lifting the arms, with palpitations, coldness of the limbs and body, and dreadful, weak feeling in pit of stomach; sensation as if the heart would stop beating if she moved, with fear of impending death; urine scanty and high colored, with brick-dust sediment.

*Lachesis*, from its effect upon the blood, and its capability of producing endocarditis, should be our most efficient remedy when the inflammation results from septicæmia, provided always that the symptoms correspond. Like the other serpent poisons, it has a double action, affecting both the blood and the nervous system, hence it is also of great use in removing many nervous symptoms accompanying the various cardiac diseases. *Symptoms*: Palpitation, can bear no pressure on throat or chest; numbness of the left arm, fainting, anxiety; restless, trembling; anxiety about the heart; hasty speech; suffocation on lying down; weight on the chest; heart feels constricted;



pulse small, weak and accelerated, unequal in volume, intermittent; hot flashes; worse after sleeping.

*Lycopus virg.* promises to be of great service, particularly in the sub-acute and chronic forms. We should know more of it. Among its symptoms are: constricting pain and tenderness around the heart; cough with spitting of blood; intermission in the beating of the heart; first sound replaced by a blowing sound at the apex, from mitral regurgitation; cardiac distress most marked at apex, on awaking and after slight exertion; pulse feeble, compressible; faintness.

*Naja trip.*—While this is similar in its action to Lachesis, it seems to spend more of its force upon the nervous system. Still, it should be useful in endocarditis resulting from blood poison. Intense dyspnoea, violent cough, frothy expectoration, with palpitation; feeling of depression and lowness about the heart, acute pain and oppression in the chest; cannot lie on the left side.

*Spigelia.*—Bayes speaks very highly of this remedy in endocarditis of rheumatic origin. It has palpitation, violent, worse bending forward; palpitation when sitting down after rising in the morning, or from the least motion; tremulous sensation in chest and temples, increased by motion; stitches in the cardiac region; cutting pains from the heart to the shoulders, as far as the head and arms; systolic blowing at the apex.

*Sulphur* should never be forgotten. Given as the inflammation begins to assume the sub-acute form, or

even in the chronic stage, it will often be of great assistance.

As it is the strain upon the valves from their constant action, which renders them almost, if not entirely incapable of being restored to their proper shape when once distorted by inflammation, and as every exertion tends to quicken the circulation and increase the violence and rapidity with which they are opened and closed, it is obvious that the patient suffering from endocarditis from whatever cause, should maintain as perfect quiet as possible. It is absolutely necessary that all stimulants should be avoided.

## CHAPTER IX.

### VALVULAR DISEASES.

These may be divided functionally into obstructive and regurgitant. Specifying each according to its anatomical seat, we have the

Mitral regurgitant,	Tricuspid regurgitant,
Mitral obstructive,	Tricuspid obstructive,
Aortic regurgitant,	Pulmonic regurgitant,
Aortic obstructive,	Pulmonic obstructive.

They are more frequently met with in middle life than at any other age. Of the forty-four cases examined by Hayden after death, the average age at time of decease was 37.3 years.

In early life the mitral valve is the one most frequently affected, the disease being, in the great majority of cases, merely a chronic endocarditis with its consequent distortion of the valves, and which is the result of acute rheumatism. In later life, the semilunar valves at the aortic orifice are more frequently the subject of the lesion, no acute symptoms having been present at the beginning, the disease having come on gradually, and being the result of strain from violent and prolonged exertion, from alcoholic stimulants, from an obstructed circulation, or from two or more of these causes combined.

As to the relative frequency of valves affected, Wil-

ligk found thirty-six cases of mitral disease to twenty-two of aortic, eight of tricuspid, and two pulmonic. Flint found forty cases of mitral disease to thirty-seven of aortic, fourteen of both combined, and four of tricuspid. Of the forty-four cases examined by Hayden after death, and tabulated by him, the aortic valves were affected in twenty-six cases, the mitral in twenty-nine, the pulmonic in one, (cancerous), and the tricuspid in five. The aortic and mitral valves both participated in eight cases.

Statistics taken from different populations show somewhat different results. Thus, in an agricultural or pastoral community, mitral disease will be found to very largely predominate, while in mining, manufacturing and commercial populations, the cases of aortic disease may sometimes be found even to considerably exceed those of mitral disease. Taken as a whole, the figures above given will probably come very near expressing their relative frequency.

The following expresses, at least approximately, the order of frequency of the varieties of lesion in each respective valve.

1. Mitral regurgitant. 2. Aortic obstructive. 3. Aortic regurgitant. 4. Mitral obstructive. 5. Tricuspid regurgitant. 6. Pulmonic obstructive. 7. Tricuspid obstructive. 8. Pulmonic regurgitant.

There are also various combinations, of which the following are some of the most frequent.

1. Aortic obstructive and aortic regurgitant.
2. Mitral obstructive and mitral regurgitant.



3. Aortic obstructive and mitral regurgitant.
4. Mitral obstructive and tricuspid regurgitant.
5. Aortic regurgitant and mitral regurgitant.
6. Mitral regurgitant and tricuspid regurgitant.
7. Aortic obstructive or regurgitant, mitral regurgitant, and tricuspid regurgitant.

The left side is more frequently affected than the right, the proportion being as about ten to one.

Men, in consequence of their greater exposure to extremes of temperature, their more violent exertions, and their more frequent indulgence in intoxicating liquors, are, in general, more apt to be affected than women.

*Mitral disease* may then be classified into obstructive and regurgitant.

*Mitral obstruction* is generally the result of rheumatic inflammation, and is a disease rather of early life than of middle age or senility. According to a recent French monograph, about seven-tenths of all those thus affected are females. The fibrous ring surrounding the opening may itself be the seat of the disease, narrowing it so as to produce obstruction to the blood stream, without any actual valve disease being present, or what is more frequently the case, the free edges of the valves may be thickened and agglutinated together, forming a firm diaphragm with a slit in the centre, the "button-hole mitral." Occasionally this curtain projects forward into the ventricle; sometimes by contraction of its tendinous attachments, sometimes by the force with which the hypertrophied auricle drives

the blood against it. It then becomes what is called a "funnel shaped mitral." Perhaps one-twentieth of the cases of mitral constriction are of this latter variety.

Considered alone, the *symptoms* of mitral constriction are of but little diagnostic value, being found also in other cardiac lesions, particularly in mitral inadequacy.

One of the most constant symptoms of mitral narrowing, is the occasional expectoration of pure red blood. Short, teasing cough is commonly present, either dry or with thin, serous expectoration, with sensation of tightness in the chest, while the respiration is usually short and easily accelerated by exertion. The temperature is generally low, from deficiency of arterial blood, and the patient is easily chilled. The pulse is apt to be somewhat smaller and quicker than natural, and, as long as the left ventricle retains its integrity, generally regular, but if the patient indulge in violent exercise, we shall be likely to have more or less irregularity, palpitation, dyspnœa, and pain at the apex. Hæmoptysis and sharp pain occasionally felt at the apex are considered by Hayden "eminently suggestive of mitral obstruction."

Hæmoptysis occurring in the earlier stages merely serves to relieve the congestion of the lungs, and need excite no apprehensions of an immediately fatal result; but in the last days, when the lungs are intensely congested, it is indicative of extravasation of blood into the lung substance, commonly called pulmonary apoplexy, and a frequent complication of this lesion.

In the last stages, all the organs of the body become congested from tricuspid failure, and there is anasarca, and finally effusion into the serous cavities. Death usually takes place from pulmonary œdema.

The *physical signs* of mitral narrowing are two in number, and eminently characteristic. They are *fremitus* and *murmur*, both of which are *presystolic in time*.

Fremitus, or thrill, is the sensation of vibration communicated to the hand when placed upon the precordium, and when observed in mitral constriction, is felt most distinctly at the apex, and occurs just before the first sound and systolic impulse. It is present in about one half the cases. When observed in any other lesion, it is *never distinctly presystolic in time*. That of mitral regurgitation, when present, is synchronous with the first sound and impulse, that of pericarditis is post-diastolic and systolic, and that of ventricular aneurism diastolic or systolic.

Presystolic murmur is to the ear what presystolic fremitus is to the hand, and, like it, is produced by the blood rushing through the narrowed and roughened orifice, from the auricle into the ventricle. Like it, also, it immediately precedes the impulse and first sound, and has its point of maximum intensity at the apex. It is pathognomonic of mitral narrowing, never being heard in any other lesion, and is all but universally present in this. If ever absent, it is in consequence of debility of the heart walls, and this debility, according to Hayden, is generally that of



impending dissolution. This murmur may be simulated by others, but a careful attention to rhythm will serve to distinguish it. It is harsh in quality, and may be represented thus: r-r-r-r; or, taken in connection with the first sound; r-r-r-up, r-r-r-up. If, however, there be mitral regurgitation, the sound will be somewhat modified by the absence of the valvular element of the first sound, and the substitution of the soft systolic apex murmur of regurgitation. The two murmurs will then assume a rather indistinctly double character, the two elements of which are separated from each other by the impulse and muscular element of the first sound, that which precedes the impulse being harsh in quality, and that which follows soft and blowing.

Percussion is of no value as an aid to diagnosis. If mitral obstruction be the only lesion present, the left ventricle is usually either normal in size or somewhat smaller than natural. The apex may, however, be displaced slightly upwards and to the left, by an enlarged right ventricle.

*Mitral insufficiency.*—As most cases of this lesion are the result of acute rheumatism, it most frequently has its origin before the age of forty years. The valve does not close properly, either in consequence of its segments becoming thickened and contracted upon themselves, and thus drawn apart; by the giving way of one or more tendinous cords, allowing one segment to float back into the auricle; by the contraction of a papillary muscle or of the tendinous cords, drawing



one or both segments forward into the ventricle; or by the yielding of the ventricular wall at the origin of a papillary muscle. Primary insufficiency is generally induced by one of these methods, and is usually the result of rheumatic endocarditis. Much less frequently, mitral insufficiency is secondary to disease at the aortic orifice. In this case, it may result from chronic inflammation from violent closure of the valve by a hypertrophied left ventricle, or from the left auriculo-ventricular opening having participated in the dilatation of the left ventricle, rendering an otherwise healthy valve incompetent to close the enlarged orifice; but more frequently these two causes are found existing together.

It may also arise from rupture of one of the valve segments. This is a rare accident, and hardly possible unless the ruptured portion has been previously diseased. Again, a tendinous cord, or portion of a valve segment, may be eaten away by ulceration in ulcerative endocarditis, and regurgitation be thus induced, or, a calcareous deposit upon the valve may have a similar result.

The older the valvular disease, the thicker and more rigid do the valve segments and their surroundings become; and this rigidity is not always fibrous alone, but, particularly in gouty constitutions, increased by calcareous deposits upon the surface, and calcification of the substance of the valve segments. In consequence of this rigidity, they offer an obstruction to the forward current, so that we very commonly

have more or less obstruction with the insufficiency, especially in the later stages.

The first important change brought about by mitral regurgitation, is enlargement of the left auricle and ventricle. The portion of blood regurgitating into the auricle, added to that coming in from the pulmonary circulation, so increases the volume of fluid to be carried forward, that increased force is required to expel it. As the auricle contracts upon its contents, the increased volume of blood over-fills the ventricle, unnaturally dilates it, and serves as a stimulus to more vigorous contraction. With the ventricular systole, a portion of the blood rushes back into the auricle with increased force, and the process is repeated as before. Thus we have the two cavities at the same time over-distending and stimulating each other, giving rise, in time, to permanent enlargement in both.

This left side enlargement in turn produces other changes, as an atheromatous condition of the lining of the left auricle and of the pulmonary veins, while the valvular disease produces a stasis of blood in the lungs.

Having thus an obstruction to the pulmonary circulation, the right ventricle becomes hypertrophied and dilated, the relation of the one to the other corresponding to the degree of perfection of nutrition. It now contracts upon its contents with more vigor than before, the tricuspid valve is closed more violently than is natural, the pulmonary artery is unduly distended, and recoils with correspondingly increased violence, closing the pulmonic semi-lunar valves more

violently than before, and producing an accented second sound at that point.

Lastly, the dilatation of the right ventricle becomes so great as to render the tricuspid valve incompetent to close the orifice, and we have all the consequences of tricuspid inadequacy and regurgitation, with venous congestion and disturbance of function in every part.

In mitral insufficiency from aortic disease, enlargement of the left ventricle precedes the mitral disease. In those cases in which mitral obstruction is marked from the beginning, the left ventricle generally retains nearly or quite its normal size, the left auricle being enlarged as the first consequence of the mitral lesion, and right side enlargement following, as in other cases of left side disease.

There are no characteristic symptoms of mitral insufficiency as distinguished from mitral obstruction. Both have the same dyspnoea, the same shortness of breath, worse on exertion, and the same teasing cough which is at first dry and afterwards may be attended with profuse frothy serous expectoration. Both have hæmoptysis, but this is much more common in obstruction than in insufficiency, the expectorated blood being generally pure, while in insufficiency it is merely a blood-streaked serum or mucus. Both are ultimately followed by the same consequences to the right heart, with general failure of the circulation, venous congestions and anasarca.

Our diagnosis then, must depend upon the physical



signs. Of these, accentuation of the pulmonic second sound, generally given so prominent a place by writers on this subject, is by no means characteristic, and may depend upon any cause which produces an obstruction to the circulation of blood in the lungs, whether it be from obstruction or regurgitation at the mitral orifice, hydrothorax, or disease within the lung itself. It may be found wherever the right ventricle is hypertrophied, provided the tricuspid valve is yet competent and the pulmonary artery still retains its elasticity.

Fremitus, although sometimes present, belongs rather to obstruction than to insufficiency.

Increased breadth of dullness from a hypertrophied left ventricle, although seeming to distinguish it from mitral obstruction, is not to be depended upon, since it might result from disease at the aortic orifice, or from any obstruction to the general circulation.

Irregularity in volume of pulse has been considered as characteristic, but it is not always present, and depends not upon the valvular disease itself, but upon the heart failure which is so often found with it.

We have left to us, then, as pathognomonic of this lesion, only its peculiar murmur. This is usually *soft and blowing* in character, but may be rasping, and is *heard loudest at the apex*. It may be diffused over a considerable portion of the left half of the thorax, particularly in young subjects. It may generally be heard at the inferior angle of the left scapula, and frequently also in the left axilla. It is *systolic in rhythm* being confined strictly to the period of contraction of



the ventricle. If, however, the left ventricle be either normal or hypertrophied, and contracting vigorously, while the regurgitation is but slight and the murmur faint, the first portion of it may be concealed by the muscular element of the first sound, which will make it appear to be post-systolic. To distinguish it from a pericardial friction sound, the reader is referred to the table of comparison in the section on pericarditis.

*Aortic valvular disease* is much more prevalent among males than females, perhaps three fourths of its victims being men. It is also most frequently met with in advanced life, although sometimes seen in youth. Unlike mitral disease, it is seldom of rheumatic origin. It consists in an inflammation of the valve segments and surroundings, generally the result of strain. The first change brought about in them is thickening. They afterwards become contracted by shrinkage of the new tissue, and generally more or less rolled up. The thickening gives rise to obstruction, while the shrinking and distortion are likely to render the valve incompetent. One or more of the diseased segments may be depressed by the pressure of the return current of blood in the aorta, or it may be ruptured or even detached from a similar cause, aided by sudden mechanical violence. In other cases, the orifice may become dilated, thus separating the valve segments. Incompetency with regurgitation will be the result of these latter changes.

The cases of obstruction are more numerous than

those of regurgitation, although the two lesions are frequently found existing together.

As disease of the aortic valves is generally atheromatous, it may be induced by anything which produces too great a strain upon them, or closes them with too great violence. Among these causes are such sports and occupations as call for violent exertion, especially with the arms, as rowing, and lifting heavy weights. Strikers at the anvil very often suffer in the same way, the heart being excited to violent action while the chest is constricted by the contraction of the muscles over it. Miners working in relays in seams of coal so thin that they cannot stand erect, frequently suffer in a similar manner.

In these cases, the left ventricle becomes hypertrophied in order to meet the increased demands upon it, and, under the stimulus of violent exertion, empties itself with great force into the aorta, which, not yet having lost its elasticity by atheroma, recoils with proportionate energy, and the valve not only becomes inflamed and thickened, but gives way before the return current, and becomes incompetent. Hence insufficiency, often associated with obstruction, is the variety of aortic lesion most frequently resulting from these several causes. It generally has its origin in youth or early adult life.

Habitual and excessive indulgence in intoxicating liquors is another and frequent cause of aortic disease. If at the same time the occupation of the individual necessitates *violent muscular exertion*, incompetency

may predominate, but if not, and especially if it occur late in life, the lesion is chiefly or entirely atheromatous, and the valve segments are thickened and rigid, and offer an obstruction to the forward current, while yet competent to prevent reflux.

The greatest cause of aortic valvular disease, as well as of atheroma in general, is the gouty diathesis. This, as is well known, is chiefly brought about by high living and sedentary habits, and is most frequently met with in commercial populations and in the aged. More or less kidney disease is generally present. The impurities of the blood produce tonic spasm of the arterioles, which obstructs the general circulation. The left ventricle then becomes hypertrophied to meet this obstruction, and, throwing the blood into the aorta with increased force, the latter vessel recoils with corresponding energy, closing the valves with unnatural violence, and giving rise to chronic inflammation and thickening, with rigidity which is increased by calcareous deposit upon the free surface, and calcareous degeneration of the products of the inflammation. The result is generally obstruction at the aortic orifice, although regurgitation is occasionally also present.

In the rigid and brittle state induced by this atheromatous process, the valve is sometimes ruptured, or one of its segments torn loose, by some violent exertion, or by leaping from a great height, and regurgitation suddenly sets in, the valve becoming hopelessly incompetent.



Sometimes, in the absence of any valve disease, a fibrinous or calcareous deposit in the orifice may give rise to obstruction or inadequacy or both, and it may happen that this will be washed away, and the murmur cease.

As consequences of aortic valvular diseases, we have further hypertrophy to counterbalance the effect of the valvular lesion, followed in the case of regurgitation without obstruction, by atheroma of the arterial system and especially of the aorta, with loss of elasticity and consequently diminished recoil, imperfect nourishment of the heart walls, dilatation and perhaps fatty degeneration.

If the lesion is purely obstructive, aortic recoil is enfeebled not merely by the atheroma, which generally accompanies but does not depend on the valvular disease, but by the diminished volume of blood thrown into the aorta, and with a similar effect upon the heart walls.

The mitral valve frequently gives way, either in consequence of chronic inflammation from violent closure by the hypertrophied ventricle, or from ventricular dilatation having rendered it incompetent; or the latter cause may be added to the former.

From the above, it will be seen that disease of the aortic valves may bring in its train all the consequences of mitral disease and of hypertrophy and dilatation. These have both been considered more fully in previous pages.

Aside from the pulse, the symptoms met with in

aortic valvular disease are of but little value in the way of diagnosis, as they usually owe their existence not to the valvular lesion, but to the changes which have taken place in the heart walls.

Commensurate with the degree of cardiac debility, we are likely to find shortness of breath, and palpitation on exertion. Pulmonary œdema and venous congestion do not appear until the mitral and tricuspid valves have respectively become incompetent.

In obstruction, there may be coldness of skin and extremities. In pure regurgitation, while ventricular contractions are still vigorous, the countenance is often flushed, and the eye sometimes presents a wild and frightened appearance, the throbbing and jarring pulse continually reminding the patient of his condition.

As long as the hypertrophy continues good, the pulse of aortic obstruction without regurgitation is apt to be *somewhat slower than natural*, steady and *prolonged*, and possesses a degree of firmness corresponding with the degree of rigidity of the arterial coats. Even then, it is liable to intermit on violent exertion, the ventricle seeming to wait to gather strength for the difficult task imposed upon it. After dilatation has made considerable progress, however, irregularity and intermittency are generally present, and increased by every exertion, although the pulse remains slow and prolonged.

When we remember that in a case of uncomplicated aortic regurgitation, at every systole of the over-filled and hypertrophied ventricle an unusually large quan-

tity of blood is suddenly thrown into the almost empty arterial system, and that while a portion of it is carried on into the capillaries, another and large portion is at once forced back again through the semi-lunar valves by the arterial and aortic recoil, we may understand what must be the character of the pulse in the early stages of that lesion. It is usually somewhat accelerated, and is full, bounding, short, and suddenly collapsing. One author has described it as feeling as if balls of blood were being shot under the finger. It is distinctly and plainly visible, not only in the carotids, but in the radial and temporal arteries. It is of so throbbing a character as often to shake the entire person.

This visible pulsation is liable to be confounded with that other pulse of unfilled arteries, which results from anæmia, especially if hypertrophy be also present with the latter condition. The pulse of atheromatous arteries is also often visible when no regurgitation is present, the vessel, in consequence of its rigidity, seeming to be partly lifted out of its bed with every pulsation. In neither of these cases does the pulse collapse so suddenly as in regurgitation, while that of anæmia is remarkable for its softness.

In regurgitation, as the arteries become more rigid, and lose their elasticity by the ever increasing atheroma, the pulse does not collapse so readily as in the early stages, and the sphygmographic tracing shows a more prolonged summit. Ventricular dilatation renders it irregular and intermitting, and more so



upon exertion. The aorta, generally greatly dilated and atheromatous, can be distinctly felt, pulsating in the sternal notch.

The two kinds of pulse above given as distinctive of aortic valvular disease, represent the extremes of the two forms of that lesion, but when obstruction and regurgitation are combined, we may have all grades between these two extremes, according as one or the other lesion predominates; and they may be so evenly balanced as to render the pulse nearly or quite natural.

*Physical Signs.*—In cases of *obstruction*, inspection and palpation show the apex displaced considerably to the left, while in *insufficiency* the displacement has been made to take more of a downward direction by the direct force of the return current. In both, except in the later stages, the heart is found to pulsate with an energy commensurate with its increased size.

Percussion shows the area of dullness increased to the left, or downwards and to the left, according as obstruction or insufficiency predominates.

Our chief reliance in making a diagnosis must be upon auscultation, the only unmistakable sign of either lesion being its accompanying murmur. This may be systolic or diastolic, single or double.

A single *systolic* murmur, with its maximum intensity in the second intercostal space at the right hand border of the sternum, and extending downwards nearly to the ensiform cartilage, *upwards along the ascending aorta and arch, and into the carotid and subclavian arteries*, and usually transmitted as far as

the apex, but *always loudest at the first named point*, is indicative of aortic obstruction or stenosis. If very loud, it may be transmitted still farther along the course of the arteries.

An aortic obstructive murmur is usually harsh in quality, and sometimes rasping or even musical. If the ventricular walls have become debilitated by degeneration, or if the mitral and tricuspid valves are incompetent, it may obscure or entirely replace the first sound of the heart.

It may be readily confounded with hæmic murmur, but may be distinguished from it when we remember that the latter is soft and blowing in quality, that it usually ceases to be heard in the carotids when the patient assumes the recumbent posture, and that it is most always attended by a continual venous hum in the jugulars. The age and sex of the patient also furnish distinguishing features, the hæmic murmur generally being found in young females, while those in whom the aortic obstructive murmur is present, are usually men somewhat advanced in years. If it be obstructive, the smaller arteries will generally be found to be atheromatous from the beginning, which is not true of the hæmic murmur.

Murmur due to aortic aneurism in this region would have a somewhat higher seat.

Calcareous deposit upon the anterior segment of the mitral valve, close to the aortic orifice, may produce a murmur exactly similar to that of aortic steno-

sis, but such a cause for confusion is so rare an occurrence as scarcely to be worthy of mention.

Fremitus is an occasional accompaniment, and when transmitted into the carotids, even though the murmur has not been detected, aortic obstruction is almost certain to be present.

*Aortic insufficiency* is distinguished from all other lesions by the presence of a murmur which is *diastolic in time*, or occurs at the moment of the beginning of expansion in the ventricle. This murmur is produced by the blood rushing back into the ventricle, the aortic recoil being the propelling power. It is in almost every instance *soft and blowing, and is not transmitted into the carotids*, the blood current, the medium of transmission of a systolic aortic murmur, taking at this time an opposite direction.

In a few instances, it has been known to be pre-diastolic, the valves being somewhat clumsy, and although completely closing and giving rise to a second sound, yet first permitting a slight regurgitation to take place. In this case the murmur would still be diastolic with reference to the pulmonic valves.

Lack of uniformity in the contraction of the two sides of the heart has occasionally caused it to be post diastolic. Here it would be distinguished from a pericardial friction sound by its soft and blowing character and the absence of fremitus.

From the fact of its non-transmission in consequence of the reversal of the blood current, the aortic re-



regurgitant murmur has its point of maximum intensity at mid-sternum, opposite the third costal cartilage.

A diastolic murmur occurring at the mouth of the pulmonary artery would be loudest at the left hand border of the sternum, in the second intercostal space, but it is so rare as not to be likely to lead to confusion.

*Right Side Valvular Disease.*—In intra-uterine life, the right side of the heart is much more frequently affected than the left, but with cases occurring at this period we are likely to have little to do. After that, the relative frequency is reversed.

Although *tricuspid regurgitation* is one of the most frequent consequences of left side valvular affections, actual disease of the tricuspid valve is by no means common. When present, it may be the result of acute endocarditis of the right side, of chronic inflammation induced by the forcible contraction of a hypertrophied right ventricle, or of rupture of one of its segments, or of a papillary muscle.

The consequence of disease at the tricuspid valve is generally insufficiency with regurgitation. Tricuspid regurgitation is far more frequently induced, however, by dilatation of the right ventricle, rendering an otherwise healthy valve incompetent. In such cases, murmur is not usually heard, in consequence of the feebleness of the ventricular contractions. If the inadequacy comes on suddenly, while yet the ventricle retains its integrity, there will be heard a murmur which is *systolic in time*, and has its point of maximum intensity at the *ensiform cartilage*, and is only trans-

mitted over a very narrow space. It is *soft and blowing* in quality. Another sign of this lesion is pulsation in the jugular veins and in the liver. The presence of jugular pulsation once established beyond a doubt, we know to a certainty that there is regurgitation at the tricuspid. It is occasionally simulated by throbbing of the carotids being communicated to the jugulars. We may readily distinguish between the two by making *slight* pressure upon the vein immediately above the clavicle. If the pulsation ceases, it is jugular; if not, it is carotid.

The consequences of tricuspid regurgitation have been already described, and need not be repeated in detail here. It is sufficient to say that the whole venous system becomes engorged, with venous congestions everywhere, enlargement of liver and spleen, derangement of function in every organ of the body, blue skin, anasarca, and finally effusions into the serous cavities, and death. Yet it is possible for slight regurgitation to exist without such terrible consequences following.

*Tricuspid obstruction* is extremely rare, and when present, is generally the result of inflammation and agglutination of the segments of the valve. It most frequently dates from foetal life.

It would be indicated by a *pre-systolic murmur at the ensiform cartilage, very feeble, and not transmitted*. This murmur could only be confounded with that of mitral obstruction, but the latter has its seat of maximum intensity at the apex.

Lesion of the *pulmonary* valve is rare. When found, it is generally obstructive, and of intra-uterine origin.

*Pulmonic obstruction* is usually accompanied by hypertrophy, and finally dilatation of the right ventricle, with tricuspid failure. It is marked by a systolic murmur, having its point of maximum intensity in the second left intercostal space, close to the sternum. This murmur is heard over a very limited area. From an obstructive murmur at the aortic orifice, it may be distinguished by not being heard at the right of the sternum, nor transmitted upwards into the carotids. It is apt to be harsh in quality, and is, in most cases, the result of inflammation and roughening of the valve segments.

Hæmic murmur in the same place is also systolic but is soft and blowing, and is usually attended by a similar murmur at the aortic orifice and in the carotids, and by venous hum in the jugulars.

Obstructive murmur in the pulmonary artery or its branches may be produced by other causes, as by the pressure of enlarged bronchial glands, of an aneurism, of a cancerous or other tumor, or of tubercular matter in the lungs. Murmur produced by this last means is not at all rare, and is apt to be widely transmitted, in consequence of the solidification of the surrounding parts.

*Pulmonic regurgitation* would be marked by a diastolic murmur in the left second intercostal space, close



to the sternum, and extending somewhat downwards towards the ensiform cartilage.

The *prognosis* of a case of valvular disease depends upon its previous duration, the age and evident vitality of the patient, his habits, and the present condition of the heart walls. It also varies according to the amount of the obstruction or regurgitation, and the position of the valve affected. In general terms, it may be said that the prospect improves as we pass from the tricuspid to the aortic orifice. When two or more valves are affected, the prognosis is less favorable than when only one is involved, and especially is this true when one valve failure is the result of another.

Tricuspid failure generally means speedy death, with venous congestion and dropsy.

Disease at the pulmonary orifice, although little known, certainly has a shorter distance to travel, and usually is much more speedily fatal than disease of the left side. Those so affected, seldom if ever live to old age.

Provided the impediment to the circulation is not too great, and the nutrition of the heart walls is good, those suffering with disease at the mitral orifice frequently live to a good old age, and even then die of some other disease, yet the patient possesses less resisting power than in health, and other diseases are rendered more certainly and speedily fatal. Other cases, again, progress rapidly to a fatal termination, and a period of a few weeks or months suffices to end the

patient's sufferings. Generally, the downward progress is gradual.

Either form of aortic disease is liable to be attended or terminated by apoplexy after the arteries have become brittle by atheroma. Only in aortic obstruction is sudden death likely to occur from cardiac paralysis, and even here it is the exception. The prognosis with respect to life and health is better in this than in any other form of valvular disease. Next to this we may rank aortic insufficiency.

*Treatment.*—Many cases of valvular disease have been reported as cured, and doubtless some, at least, of these supposed cures, were such in reality, but in regard to this matter, there are three principal sources of error, viz: 1. A basic murmur may have been hæmic, and ceased with the improvement in the quality of the blood. 2. An apex murmur may have been dynamic regurgitant, and ceased with return of strength to the heart walls. 3. Either a basic or an apex murmur may have been organic, and yet have ceased to be audible in consequence of extreme cardiac debility. It is a very common thing for murmurs to cease in this manner during the last few days of the patient's existence. Such cessation should be regarded as of evil omen.

It is certain that in the present state of our knowledge, very few cases of valvular disease in proportion to the whole can be cured. This will perhaps be more readily understood when we remember how difficult it is to restore to shape and usefulness, a hand withered

and distorted with chronic rheumatism. If to the distortion of a valve by the inflammatory process, be added calcareous deposits upon its surface, and calcareous degeneration within it, and the necessity for constant use of the part, the case becomes still more hopeless as far as a cure is concerned. Yet in most cases it would be well to remember that a cure is not always an absolute impossibility, and, without promising any favorable results, that it is well worth one's while to try and see what may be done to that end. If the disease is of recent origin, possibly some little, at least, of the thickening, or contraction, or other distortion, may be removed; or, in an old case, it might be that by a favorable change in the nutrition, the valve could be made to shed all or a portion of the calcareous deposit upon it, in some such way as old finger and toe nails are sometimes shed in the process of cure of long standing disease. In either case, it is possible that the change thus brought about may be sufficient to restore the normal functions of the valve. When this is not possible, no doubt, in many cases, further distortion may be arrested by timely interference.

But, granting that in most cases little or no change can be made in the distorted valve, still the treatment can be undertaken with good reason for encouragement, for in nearly every case the patient can be rendered much more comfortable, and, except in the last stages, his life can generally be prolonged. Sometimes, indeed, by the use of judiciously selected reme-



dies, and the proper attention to hygiene, many years are added.

The object of treatment in a case of valvular disease should be:

1. To prevent further distortion of the valves.
2. To restore or maintain the integrity of the heart walls, removing or preventing dilatation as far as possible.
3. To remove those inconveniences which the general system suffers, in consequence of the obstructed circulation.

1. To the accomplishment of the first named object, *Arnica* or *Rhus tox.* may be found beneficial, if the valvular disease be the result of over-exertion. They will find their chief use in aortic disease, but may be indicated in mitral also. Possibly *Plumbum* may do something when it is a consequence of Bright's disease. *Sulphur*, *Cactus*, *Rhus tox.*, *Naja trip.*, and *Lachesis* might be studied with profit in mitral disease; the first three especially if it is of rheumatic origin. *Lycopus virg.* also promises excellent results in this direction. (For further indications, see the treatment of endocarditis).

2. For the removal of the accompanying dilatation, see the remedies under the head of hypertrophy. Also study the materia medica, for in these cases there is always ample time to select the remedy with care. *Digitalis* is of immense value, always to be used according to indications, and not indiscriminately. Other

important remedies are Aurum, Lachesis, Lycopus, Nux vom., Spigelia and Spongia.

3. The consequences of an obstructed circulation are, speaking in a general way, best removed by restoring, as far as possible, the vigor of the heart walls, according to the last section.

For the terrible cardiac asthma which is so common, Spongia is often of great use, especially if the patient is compelled to sit up during the after part of the night. He wakes with fright and fear of suffocation. If given without regard to indications, disappointment is pretty apt to follow.

The gastric disorders which nearly always accompany the later stages of valvular disease, after the right side has become affected, generally result from venous congestion which is the consequence of heart failure, and this fact must be taken into consideration in making choice of the remedy. The same is true of many other derangements occurring at this period.

Digitalis is perhaps the most efficient remedy for the anasarca of the later stages, not only relieving the pressure upon the walls of the veins, but increasing arterial tension in the kidneys by strengthening the heart walls. Lycopodium should not be forgotten.

When the internal remedy is no longer capable of doing the work, puncturing the skin of the swollen extremities carries off the extravasated serum, and thus affords relief to the patient. This relief is of course of short duration, but it is better than none. The principal objection to it is the liability of erysip-

elas or gangrene to set in at the places where the openings are made. Old School physicians frequently use such purgatives as Elaterium with apparent benefit, as a considerable quantity of water may be carried off in this way, but this not only has the disadvantage of debilitating the patient, but in the last stages is much less efficient than the puncture.

The digestion and nutrition should be kept as perfect as possible. The importance of this cannot be over-estimated. Whenever the patient's strength is sufficient to allow it, moderate exercise in the open air should be insisted on. If his calling is one which requires no violent exertion or excitement, it is better for him to continue to follow it in moderation, than to devote himself entirely to the care of his health, and think of nothing but himself and his disease.



## CHAPTER X.

### EMBOLISM.

An occasional consequence of endocarditis, as also of chronic valvular disease, is embolism. A portion of the deposit upon a valve, whether fibrinous or calcareous, but generally the former, becomes washed loose in the blood stream, and is carried on until the artery becomes too narrow for its further passage. Here it lodges, and blocks up the vessel as a plug, cutting off the circulation, and producing stasis of blood in the vessels of the part beyond.

The embolus generally springs from the left side of the heart, and usually from the mitral valve. It may lodge in almost any part of the body, but most frequently in the kidneys, next in the spleen, and next perhaps in the brain, while other parts, as the skin and extremities, rarely suffer.

If a sufficient collateral circulation is soon established, but little harm may result; but if the circulation be completely cut off, as sometimes is the case in the fingers and toes, gangrene will be likely to ensue. If the affected tract be small, or if the embolus results from ulcerative endocarditis, we may have abscess. In many cases the plug becomes dissolved and the circulation in the part nearly or quite restored.

If it takes an upward direction, it generally lodges

in the middle cerebral artery, and much more frequently the left than the right. The result is paralysis of the opposite side, but this paralysis is one of motion merely, the sensation being either natural or exalted. From the nature and seat of the affection, it must generally end in death, but should the plug be dissolved, partial or entire recovery may take place.

Emboli have been known to lodge in the *arteria centralis retinae*, loss of sight in the affected eye of course resulting.

An embolus lodging in the pulmonary artery or one of its branches, may have started from a vein as the result of inflammation there, or from the valves of the right side of the heart. It is more or less dangerous according to the extent of the tract from which the circulation is cut off, and the condition of the opposite lung. Death may ensue within a few minutes or hours, or a partial recovery may take place. The symptoms are those of thrombus, described among the terminations of dilatation.

*Treatment.*—All that the physician can do is likely to be of little if any avail in affecting the ultimate result. The patient's condition can best be ameliorated by the remedy which bears the closest Homœopathic relation to the symptoms of the case. In choosing this, we should consider not only the organ affected, but the nature of the resulting pathological condition.

## CHAPTER XI.

### NEUROSIS. NERVOUS PALPITATION.

Palpitation consists in a more or less irregular and excited action of the heart, accompanied by a sensation of smothering or other distress. It should be taken as an evidence of over-distention of the cavities of the heart. It is not a sign of power, but of debility. It is the struggle which that organ makes in order to expel its contents when its walls are debilitated, or when the blood stream is in any manner obstructed.

The debility may be either the result of derangement of the nervous system, or of actual disease of the heart walls.

The obstruction may be continuous, or only momentary; it may be mechanical, as from chronic valvular disease, from the contraction of the muscles across the arteries during exertion, or from a distended stomach pressing upon the aorta, when lying upon the back, or upon the heart itself when stooping; or it may be of nervous origin, as in derangement of the sympathetic, where the capillaries and the smaller arteries of the systemic circulation are spasmodically contracted. An example of this is found in hysterical palpitation, and here we have as a consequence, cool skin with increased pressure upon the kidneys and increased quantity of urine.



Derangement of the sympathetic with palpitation, is likely to occur at puberty, at the climacteric, and after too great indulgence of any of the appetites and passions. It may also be found in connection with disease of any organ of the body, but more frequently with gastric or ovarian disorders. It very frequently results also from sudden emotion, as fright, grief, or joy.

Palpitation resulting from organic disease is worse upon exertion, which is not true of that which is of nervous origin. Nervous palpitation, on the other hand, is much more likely to attract the attention and excite the fears of the patient. It is most frequently met with in the young and those of middle age. Women, in consequence of their peculiarities of constitution, are far more frequently affected than men.

The *prognosis* depends upon the circumstances under which it is found. If nervous palpitation accompanies organic heart disease, the patient may be in a very dangerous condition, but when not so associated, it is in most cases readily curable.

*Treatment.*—When it depends upon debility of the heart wall itself, as in dilitation, such remedies as Cactus, Digitalis, Lachesis, Lycopodium and Nux vom. will be found useful, according to their respective indications, as given below, and also under the head of hypertrophy.

If it results from anæmia, China, Cuprum, Ferrum, Ignatia or Pulsatilla will perhaps be indicated, and most likely cure the case.

If from disorders of the female sexual system, give whatever seems to be indicated by the totality of the symptoms of that affection, always keeping in view the palpitation as one of the symptoms. Among the most frequently indicated, will be China, Lachesis, Lilium, Nux vomica, Pulsatilla and Sepia.

If disorders of the digestive sphere be the cause, the remedy may be found among such medicines as Lycopodium, Nux vom., Pulsatilla or Sulphur.

If of emotional origin, Aconite, Badiaga, Coffea, Ignatia or Nux vom.

*Aconite*.—In young and plethoric subjects; after fright; after wine; palpitation with feeling as if boiling water was poured into the chest, fainting with tingling in fingers, numbness and lameness of left arm, fear of death; anxiety, difficulty of breathing, flying heat in the face, sensation as of something rushing into the head.

*Agaricus*.—Violent palpitation, worse in the evening, with redness of the face; constant feeling as of a lump in the epigastrium, with pain under the sternum; drunkards.

*Amyl nitrate*.—Cardiac oppression and tumultuous heart's action; violent pulsation of carotids, extending to head and temples, with flushing of the face, followed by sweat.

*Arsenicum*.—Palpitation, after suppressed herpes or foot-sweat; palpitation with small, irregular pulse; worse at night; palpitation with tremulous weakness after stool; pulse rapid and weak, or small, very

weak and irregular. Dropsical tendency; chronic diarrhœa.

*Aurum*.—Violent beating of the heart after exertion, with great agony, frequent attacks of anguish about the heart, with tremulous fearfulness; when riding or walking, palpitation compels to stop; palpitation with irregular intermittent pulse, and strong breathing; despondency, disposition to suicide. Syphilitic subjects; after mercury.

*Badiaga*.—Nervous palpitations, with cardiac debility; palpitation from any elating thought, with a fluttering and vibrating upon the slightest emotion of the mind.

*Cactus grand.*—Palpitation of the heart, continues day and night, worse when walking and when lying on the left side; the palpitation consists of small irregular beats, with necessity for deep inspiration; slight excitement or deep thought is sufficient to produce this condition; sensation of constriction in the heart, as if an iron band prevented its normal movement; violent palpitation, in upper part of chest, at night in bed.

*China*.—Nervous palpitations after great loss of fluids, or prolonged nursing; heat and redness of the face, with cold hands; nightly diarrhœa, with large, undigested, light colored stools.

*Cocculus*.—Tremulous palpitation from quick motion or mental excitement, with dizziness and faintness.

*Digitalis*.—Violent, but not very rapid beating of



the heart, irregular and intermittent pulse, and brick-dust sediment in urine; very feeble, irregular action of the heart, with feeble, small, and intermittent pulse; sensation as if the heart would stop beating if she moved, with fear of impending death; dreadful, weak feeling in the pit of the stomach.

*Ferrum*.—Anæmia; throbbing in all the blood vessels. A soft systolic murmur at the base of the heart and extending upwards along the carotids would be an indication for this remedy, as denoting an impoverished condition of the blood.

*Lachesis*.—Palpitation, can bear no pressure on throat or chest; must sit up or lie on the right side; palpitation and choking from the slightest exertion; deep sighing every few minutes; worse after sleeping; hasty speech; hot flashes; change of life.

*Laurocerasus*.—Heart's action irregular, pulse slow; beating, fluttering sensation in the region of the heart; gasps for breath.

*Lilium tig.*—Cardiac irritability, nervous palpitation; pains dull, pressive and heavy, as if the heart were grasped and released alternately; frequent sensation as if the heart stopped, followed by a rush of blood to the heart and violent palpitation; sharp, quick pain in the left chest, with fluttering at the heart; worse when lying on either side, or *worse when lying on right side, better on left*. Prolapsus uteri; great bearing down, and sensation as if the whole pelvic contents would come out through the vagina.

*Lithium carb.*.—Nervous palpitation, cardiac irritability; valvular affections, especially in gouty cases; painfulness of feet, ankles, metatarsus and toes, especially of the borders of the feet and soles, burning in great toe.

*Lycopodium.*.—Palpitation of heart, worse in evening; indigestion, bloating of stomach and abdomen, eating only a few mouthfuls seems to fill him to repletion; rumbling in abdomen, constipation, red sand in the urine.

*Nux vomica.*.—Palpitation in frequent short paroxysms, with pulsating throbs in the direction of the heart, especially from mental emotions, protracted study, after eating highly seasoned food; heart feels tired; palpitation on lying down; frequent belching; dilatation of heart; nervous palpitation and heaviness of the chest; indigestion, acidity of stomach, constipation. People of sedentary habits.

*Pulsatilla.*.—Palpitation in young girls during the time of puberty, or from amenorrhœa; palpitation in violent paroxysms, often with anguish and obscuration of sight; trembling of limbs; anæmia; palpitation with vertigo; palpitation after dinner; greasy food disagrees, she spits grease.

*Sepia.*.—Nervous palpitation, better when walking fast; palpitation, with anxiety about things which happened years ago; palpitation after mental emotion; constipation, sense of weight at the anus; menses scanty or suppressed; badly smelling leucorrhœa, worse before menses; bearing down in uterine region,

crosses legs to prevent parts from protruding; prolapsus of uterus or vagina.

*Spigelia*.—Violent palpitation, worse bending forward; high fever; stitching pains in chest; palpitation on sitting down, after rising in the morning; palpitation from the least motion; palpitation with vertigo; rheumatic subjects; pulse irregular, strong, and slow, trembling.

*Tabacum*.—Palpitation with oppression, in attacks at night, with pain in cardiac region; heart's action very feeble; violent palpitation when lying on left side; goes off when turning to right.

Care should be taken to choose the remedy, not from the indication of palpitation alone, nor even from the palpitation as produced by a certain cause, but from the totality of the symptoms of the entire case, always giving due prominence to characteristics.

Palpitation in plethoric subjects will be greatly relieved by applications of cold water or ice to the precordium.



## CHAPTER XII.

### IRRITABLE HEART.

There is such a thing as an irritable heart, without any positive debility, nor anything else abnormal, except, in the majority of cases, a slight hypertrophy, or in a few instances, some little dilatation. It is characterized by a pulse which, though it may be nearly normal when lying down, becomes greatly accelerated on assuming the erect posture, or upon exertion.

Among the causes are the attempt to labor too soon after recovery from a debilitating disease, long and rapid marches, repeated indulgence in violent passion, intense anxiety, and too active a life generally; but oftentimes its origin is unknown.

There is generally more or less pain near the apex or in the left shoulder, with palpitation which is sometimes worse at night and sometimes after exertion. Lying on the left side usually aggravates. The pulse is very rapid, especially during motion or when standing erect, when it ranges from 100 to 140 or even higher. Respiration is proportionately increased in frequency, and there is usually more or less dyspnœa, with cough, drowsiness, headache, sleeplessness, startings and frightful dreams. After violent exertion, even hæmoptysis may occur.

Physical examination discovers nothing but an un-

naturally active and jerking pulsation of the heart. If the cause can be ascertained and removed, a cure may often be speedily effected, while other cases, which seem to have taken a deeper hold on the nervous system, may be exceedingly obstinate. It is not an especially dangerous affection.

*Treatment.*—The first thing to be done, is of course to remove the cause, as far as possible.

*Aconite* is often of considerable assistance. Pulse rapid and firm; hot and dry skin, thirst.

*Bryonia* is one of the most frequently indicated remedies, and has done much good service in this affection. It would be well to remember, however, that here at least, the single characteristic, “aggravated by motion,” is not sufficient, since this symptom is common to all cases. Oppression in region of heart; stitching pain; cramp in region of heart, aggravated by walking, raising one’s self, or using the slightest exertion; heart beats violently and rapidly; pulse full, hard and tense, at times intermittent; thirst for large quantities of water; sour, sticky night sweats; constipation.

*Digitalis* will find its place where there is evidence of cardiac debility, with feeble, irregular and intermittent pulse. It should not, however, be used lower than the third decimal attenuation.

*Iodine* has extremely rapid pulse; palpitation aggravated by even the slightest motion; symptoms generally aggravated by sitting up in bed.

*Phosphorus.*—Great pressure on the middle of the

sternum; dyspnœa, with inability to exert himself; palpitation; frequent pulse; great sensitiveness to external impressions; pulmonary affections; disease of the right heart; painless diarrhœa.

Study also *Arnica*, *Arsenicum*, *Gelsemium* and *Rhus tox*.

Should there be considerable debility, or the nervous system be disordered, in addition to the cardiac irritability, these conditions should be removed by the properly selected Homœopathic remedy.



## CHAPTER XIII.

### GRAVES' OR BASEDOW'S DISEASE—EXOPHTHALMIC

#### GOITRE.

This somewhat rare disease is characterized by the three prominent symptoms, extreme cardiac activity, protrusion of the eyeballs, and enlargement of the thyroid gland. They usually manifest themselves in the order here mentioned. One or the other may be absent without seriously interfering with the diagnosis, the remaining symptoms being sufficiently characteristic.

The cardiac excitement is generally accompanied by more or less palpitation, with throbbing of the carotids. The pulse ranges from 120 to 180 beats in the minute, and is weak and irregular.

The eyeballs are so prominent as to attract attention and give the patient a wild look, while the pupils are generally natural. The prominence seems to be due in most cases to enlargement of the posterior orbital vessels, yet occasionally the cushion of fat in this position is found greatly hypertrophied, adding to the outward pressure. In many cases the eyes cannot be closed in consequence of the great protrusion, they become dry and inflamed, and even ulceration may set in, eventually destroying the sight.

The thyroid body is greatly enlarged, the right side being the first affected. It is generally soft, elastic, and pulsating, thus differing from true goitre or bronchocele, in which the gland shows considerable firmness upon pressure. and attains to a greater size than in Graves' disease. In consequence of the pulsation, the tumor of exophthalmic goitre has been mistaken for an aneurism of the carotid. In the later stages, there is increase of connective tissue, and consequently greater solidity of the part.

The swelling of the thyroid and the prominence of the eyes are subject to paroxysms of exacerbation upon increased excitement of the nervous and arterial systems.

The essential condition in this singular affection, seems to be such a derangement of the sympathetic as to destroy the balance between the vaso-motor and vaso-inhibitory nerves, but why this should manifest itself more in the arteries of the orbit and thyroid than elsewhere, is not yet known. A necessary requirement seems to be a highly excitable emotional temperament. For this reason, about nine-tenths of all the cases are women.

The disease usually first manifests itself between the ages of fifteen and thirty. In addition to the three prominent symptoms already mentioned, the apex of the heart is often slightly displaced upwards and to the left by simple dilatation, which is usually present in greater or less degree, while a systolic apex murmur is occasionally heard from adynamic mitral re-

gurgitation. The anæmia which is probably always present, may produce also a *soft* systolic murmur at the aortic orifice, and there may be venous hum in the jugulars. Thrill in the thyroid is often present during periods of excitement.

The pressure of the enlarged gland is likely to produce wheezing and hoarseness, and sometimes even threatens suffocation. In females, leucorrhœa and amenorrhœa are common.

The *etiology* of this disease is little understood. Its first manifestation is usually after some severe shock, some violent emotion or terrible anxiety. It may be either acute or chronic, coming on either suddenly or gradually. Although usually beginning before the age of thirty, it seldom ceases until after the menopause, and frequently continues as long as life lasts. It is not a dangerous disease, inasmuch as it seldom produces death. In those who die while suffering with it, death is generally traceable to some other cause. When it does terminate fatally, it is usually through heart failure.

The *diagnosis* is easy, the presence of any two of the characteristic symptoms being a sufficient indication of the nature of the disease.

The *treatment* is not as satisfactory as could be wished. The disease is seldom fatal, and has a decided tendency to terminate spontaneously, especially after the climacteric, and for this reason, no matter how much our remedies may have done for the patient, we cannot be certain that nature has not done the work



unaided after all. Our self-styled "regular" friends acknowledge their want of success, and substantially admit that those of their remedies which, apparently to them, ought to cure it, although not without effect, are either positively injurious or only temporarily beneficial.

In selecting the remedy, let us bear in mind that the affection with which we have to deal is most probably a pure neurosis. The medicine to be given, once selected, should be allowed a considerable length of time to act, for here at least, nothing can be gained by frequent changes.

*Arsenicum*.—Wild look; protruding eyes; eyeballs protruding, as if out of their sockets; pupils contracted or dilated; pulse very frequent, weak and irregular; painless swelling of the external neck; swelling of the thyroid gland, especially on the right side of the neck; thirst for small quantities of water, frequently repeated; suppressed herpetic eruptions; tendency to general dropsy; debility; loss of flesh.

*Belladonna*.—Eye-balls protruding, pupils dilated, staring look; eye-balls projecting and very sparkling; face flushed; vertigo especially when stooping, with flickering before the eyes, and anguish; violent palpitation of the heart. Cerebral symptoms prominent.

*Iodium*.—Eyes glistening, injected, protruding; inflamed eyes; obstructed respiration; constriction of chest; palpitation of heart increased by movement; pulse rapid, small, and sometimes irregular or intermittent, quickened by movement; goitre, swelling of

cervical glands; coldness of hands and feet, rapid failing of strength, emaciation, sleeplessness.

*Lycopus virg.*—Eyes full, heavy and pressing outwards; dull pain in supra-orbital region; sensation of constriction in cardiac region; systolic murmur at apex (dynamic); pulse generally somewhat accelerated; oppressed respiration with sighing; rheumatic pains in various parts.

*Natrum mur.*—Redness of the white of the eye with lachrymation; eyes give out on using them; sensation of constriction in heart, as if it had not room to expand; painful stitch in the heart after reading aloud; palpitation, worse going up stairs; intermission in the beating of the heart; pulse rapid and intermittent; beating over whole body; very much inclined to weep; great depression, melancholy; disinclined to mental work.

*Spongia.*—Eyes protruding and staring, or deeply sunken; the region of the thyroid gland seems indurated; hoarseness, dry cough, difficult respiration; palpitation of the heart, rush of blood to the chest; pulse increased in fullness and rapidity.

*Veratrum album.*—Eyes distorted, protruding, surrounded by blue or black rings; sensation of dryness of the lids; pupils dilated or contracted; double vision; paroxysms of constriction of the glottis, with protruding eyes; seems in danger of suffocation, the respiration is so restricted; palpitation with anxiety, and rapid, audible respirations; pulse very rapid or

very slow; pulse very feeble; coldness of the extremities; nails blue; extreme debility.

Dr. J. S. Mitchell reports a case cured with *Arsenicum*.

Study also, Baryta carb. Calcareo carb. Digitalis and Phosphorus. Dr. R. Ludlam reports the case of a woman, in whom Spongia 3 restored the menses in a few days, and in four weeks had greatly improved the case in all other respects.

Dr. Kirsch reports five cases cured with Veratrum. Our literature on the subject is very meager.



## CHAPTER XIV.

### ANGINA PECTORIS.

Hardly any two authors agree as to the precise nature of this distressing affection; hence, it is known under different names, as *angina pectoris*, *cardiac myalgia*; *neuralgia of the heart*, etc., each of which is an attempt to express the nature of the malady.

In the vast majority of cases, it is an accompaniment of some organic lesion of the heart; most frequently, perhaps, calcification of the coronary arteries and fatty degeneration. *Atheroma* of the aorta and the gouty diathesis seem to be favorable conditions for its development. It seldom occurs before the age of forty-five, and perhaps seven-eighths of the cases are in persons over fifty years of age. Of eighty-eight cases summarized by Forbes, eighty were in men. Those cases occurring in women, most frequently originate at the climacteric. It thus attacks by preference, the aged, the male sex, and those occupying the higher walks of life. Its cause is unknown. It has been thought by some to be dependent upon calcification of the coronary arteries, but, in the majority of cases, there is no evidence that this structural change has taken place. It may even occur when no cardiac disease exists, and in persons otherwise apparently healthy.

The exciting cause is apt to be walking against the wind, getting into a cold bed, a violent fit of passion, or vigorous exercise, especially after a full meal. At other times, it may occur during the night, or when otherwise perfectly quiet.

A violent and constrictive pain is suddenly felt shooting across the cardiac region, up to the shoulder and down one or both arms, but most frequently the left. The heart's action may be normal, but is generally more or less accelerated, irregular, and so weak as to be almost imperceptible; the pulse is correspondingly rapid and feeble, the face is pale, with an appearance of great alarm and intense and generally indescribable agony, while great drops of sweat are seen coursing down the forehead and temples. The patient is usually in a fixed position, erect or slightly bent, and cannot be induced to move. The respiration is exceedingly shallow, anxious, and often rapid, yet there is no true dyspnœa, for when an effort is finally made, the air freely enters every part of the lungs. Palpitation is not present. The attack may last thus from a few seconds to an hour or longer, and generally passes off with eructations of gas, and frequently with vomiting or the emission of pale watery urine. It may recur in a few days or weeks, or not for months or even years; or, where there is no organic disease, it may never return. The paroxysms usually become more frequent with advancing years, and the progress of the accompanying disease, so that in the later stages,

several paroxysms have been known to occur in a single day.

In fatal cases, death probably results from paralysis of a heart already weakened by disease. This may, as has been suggested, be the result of spasm of the coronary arteries and their branches, cutting off the blood supply. In a somewhat similar manner, it is no unusual occurrence for other parts to become pale, cold, and partially paralyzed, when the seat of severe neuralgic pain.

Angina pectoris may be distinguished from cardiac asthma, by the fact that in the latter, the lips are blue and the breathing labored, while in the former these symptoms are not present, the respiration being short, quick and anxious, but *without effort*.

It might also be confounded with hysterical asthma, sometimes met with in young females, but may be distinguished from it by the *absence of pain* in the latter.

Angina pectoris is a rare disease, and there are not many physicians engaged in general practice, who can truthfully say that they have met with half a dozen cases in a lifetime. Jahr, in his forty years of practice, met but one.

In the majority of cases, the prognosis is decidedly unfavorable as to the ultimate result. This depends, however, upon the cause and accompanying conditions.

If the angina is an accompaniment of some organic lesion of the heart and arteries, the patient may die in the first paroxysm, or may live through many, which become more serious as the organic disease pro-



gresses, until finally an attack proves fatal, or death takes place as a result of the organic disease. Perhaps three-fourths of those who are subject to it die suddenly during a paroxysm. In those cases in which no organic disease is present, it is certainly as curable as any neuralgia or other nervous affection, while in complicated cases, much may be done to diminish the frequency of the paroxysms by the administration, during the intervals, of medicines chosen with reference not merely to the angina, but to the accompanying organic disease.

In all cases, the patient should be thoroughly examined in order to ascertain if the signs of organic disease be present.

*Treatment.*—The objects of treatment are to lessen the severity and duration of the paroxysm, and to prevent its recurrence.

However probable it may be that many attacks have been shortened by the use of the properly chosen remedy, its extremely short duration, even when no effort has been made to relieve, renders it impossible to say positively that such results have been obtained by any system of treatment. Notwithstanding this fact, the patient's sufferings are so acute that the physician who, being called during an attack, should fail to make some effort for his relief, would be guilty of gross neglect.

Amyl nitrite, Chloral hydrate, mustard plasters, and the hypodermic injection of Morphia; these are the principal means employed by Old School practition-

ers, yet each of these has men of standing and ability opposed to it, on the ground of the extreme danger attending its use in a disease at once so serious and so little understood.

The urgency of the case and the anxiety of friends, demand that something be given without delay, and for this reason, there is little opportunity for a careful selection of the remedy during the paroxysm, but the conscientious and wide awake physician will not fail to study the case afterwards and endeavor to find the true similitum.

*Aconite*.—Especially useful in recent cases, occurring in young and plethoric subjects, and from exposure to cold dry winds. Intense anxiety with fear of death. Fear of suffocation; dreadful oppression in the precordial region; cold sweat, respiration quick and anxious, intense oppressive pain in region of the heart; anxiety in cardiac region, and oppression of chest, with contracted pulse and constriction of the chest, when sitting after much motion.

*Amyl nitrite*.—Flushing of face. Choking feeling in throat, on each side of the trachea, along the carotids; feeling of constriction; the feeling of constriction in the throat extended to chest, and produced dyspnoea and asthmatic feeling in larynx and trachea, with desire to eructate; precordial anxiety; heart's action increased and often tumultuous; aching pain and constriction around the heart; general relaxation, weak feeling over the whole body.

*Arnica*.—Pain in region of heart, as if it were

squeezed together, or as if it had got a shock; stitches in the cardiac region; stitches in the heart from the left side to the right; region of base of heart feels bruised; pulse feeble, hurried and irregular; short and panting respiration. Especially indicated if the attack can be traced to any kind of violent exertion.

*Arsenicum alb.*—Frequent oppressive shortness of breath, in every position of the body, causing anxiety; cramp in the heart; quick, weak and irregular pulse, sometimes intermitting, or scarcely perceptible; apprehension, constant dread of death. *Concomitants.* Dropsy; renal affections; cardiac or idiopathic asthma; malarial poisoning; herpetic eruptions.

*Arsenicum iod.*—Great pain in cardiac region, going through to the back, in hypertrophy of the left ventricle. (Lilienthal.)

*Aurum.*—Disgust for life, suicidal tendency; despondent, melancholy; anguish increasing unto self-destruction; great anguish, coming from the precordial region, and driving him from place to place, so that he can remain nowhere; frequent attacks of anguish about the heart, with tremulous fearfulness; dyspnoea with dull stitches in the chest, when inspiring; suffocative fits, with spasmodic constriction of the chest. *Concomitants.*—Organic affection of the heart; syphilis; hydrargyrosis.

*Cactus grand.*—Sanguineous congestion in the chest, which prevents him from lying down in bed; sensation of constriction in the heart, as if an iron band prevented its normal movement; very acute pain, and



such painful stitches in the heart as to cause him to weep and cry out loudly; periodical attacks of suffocation, with fainting, cold perspiration on the face, and loss of pulse.

*Digitalis*.—Suffocative, painful constriction of the chest, as if the internal parts were grown together, especially in the morning on waking; he is obliged to quickly sit upright; a sudden sensation as though the heart stood still, with great anxiety, and necessity for holding the breath. The heart's action has lost its force; its beats are more frequent and intermittent, or slower and intermittent, and sometimes irregular; scarcely perceptible beating of the heart. Especially useful where there is marked cardiac debility, as in dilatation; cardiac dropsy, with scanty and turbid urine of high specific gravity.

*Dioscorea*.—Pain extending from chest to both arms and hands. (Lilienthal.) Sharp pain and faint feeling at region of heart; sharp pain in region of heart, arresting motions; sharp and darting pain in region of heart, arresting breathing.

*Lachesis trigon*.—Desperate fits of suffocation, must sit up in bed; feeling of constriction about the heart; pain in the precordial region causing palpitation with anxiety; she feels the beating of the heart, with weakness even to sinking down; stretching of the limbs, and a feeling about the heart as if it were constricted. The attack comes on after sleeping.

*Lactuca virosa*.—She feared to take a deep breath on account of tightness of the left chest, there was

then felt a slight shock; sometimes awakened from sleep at night on account of great tightness of the chest, so that he starts up anxiously; cramping pressive pains in various parts of the chest.

*Lycopodium*.—Dyspnœa, as though the chest were constricted by cramp; constrictive pain in the middle of the chest, from walking in the open air; oppression of the chest as if too full; acceleration of the pulse, with coldness of the face and feet. Constipation, flatulence, sensation of repletion even after eating only a few mouthfuls.

*Oxalic acid*.—Pain in the heart, soreness, stitches from behind forwards and from above downward; sharp, darting pain in the heart and left lung, extending down to the epigastrium; angina pectoris; numbness from the shoulders to the tips of the fingers.

*Phytolacca dec.*—Shocks of pain in cardiac region; pain goes into right arm; heart's action weak with constipation; pulse small and irregular, with great excitement in chest, especially in cardiac region.

*Rhus tox.*—Valuable in some cases in which there is a marked rheumatic diathesis; especially if the attacks are brought on by getting wet. General restlessness; stiffness and lameness of all the joints.

*Spigelia*.—Cutting constriction in the chest, with anxiety; dull stitch in the left side, just beneath the heart; dull sticking at the apex; recurrent dull stitches at the apex, or rather more externally.

*Tabacum*.—Palpitation in attacks at night; tight across the chest, with angina pectoris; pain between

the shoulders in angina pectoris; sudden precordial anguish.

*Tarantula*.—Great oppression of the chest; panting respiration, palpitation and prostration; oppression and panting respiration at the slightest movement; precordial anxiety, tumultuous beating of the heart; violent anguish of the heart; pain in the heart as if squeezed or pressed, also in the aorta, under the clavicle, and in the carotids, with violent throbbing of those arteries and of the heart.

Perhaps in a majority of cases Aconite will be found to correspond more nearly than any other remedy, to the totality of the symptoms of the paroxysm, and if called during an attack, I should prefer to begin the treatment with this remedy until some other is found to be better indicated. Its greatest use, however, will be during the paroxysms, after which, in many cases, some other remedy must be chosen, to prevent a recurrence. Aconite should be used in the first attenuation, or tincture, in water, a dose every five or ten minutes until the patient is relieved.

The similarity of Amyl nitrite to angina, it seems to me is more apparent than real.

Arsenicum is one of our best remedies for this affection. Both it and Spigelia I should expect to act better when given as high as the thirtieth.

If the patient is a female, passing through the climacteric, and subject to frequent flashes of heat, Lachesis is most likely to be the remedy; yet it should



not be forgotten in cases of organic disease, for here it is also a remedy of great importance.

Hot fomentations, hot foot baths and mustard plasters may be a temporary benefit to the *physician*, by consuming a little time, and convincing the friends that he takes an active interest in the patient's welfare, but they certainly can do but little to relieve the agony, and are likely to lead to confusion. In other affections, however severe, their beneficial effects upon the patient are nothing when compared with that to be derived from the administration of the properly selected Homœopathic remedy, and why should this be less true in angina pectoris? No matter how intense the agony, nor how dangerous the disease, it has not been my custom to make much use of these means, for I hold that the more serious the affection, the more necessary does it become to know if our remedy is having its desired effect.

In most cases, the proper remedy to be given during the paroxysms, is the one best calculated to prevent its recurrence.

Plumbum has not been mentioned here, because I cannot find in its provings any symptoms resembling those of angina, but it should not be forgotten as a most valuable remedy in one of the leading conditions favorable to the development of that affection, viz: combined heart and kidney disease.

The following cases may serve to illustrate, in a measure, the action of our remedies.

I. A middle-aged lady, being suddenly awakened at

night with the announcement that her child was dying, was seized with a paroxysm of angina, which was followed by a similar attack every night at the same hour. This was continued for three weeks, during all of which time her Old School physician administered powerful "tonics" with no other effect than to aggravate the malady. Dr. E. M. Hale being then called, gave Aconite 1x in a glass of water, a spoonful every two hours. She had a slight attack the first night, none afterward, and in a week was so much improved that it only required a few doses of Ignatia 30x to restore completely the tone of her nervous system.

II. Dr. T. C. Duncan was called upon to prescribe for a man who had for some time been subject to angina, the attacks occurring every two or three days. There was much pain, and the characteristic constriction being present, *Cactus grand.* was given, with the effect of so far improving the case that there was no return of the paroxysms for at least several months, at the end of which time the patient was lost sight of.

III. A lady aged forty-two, with marked symptoms of fatty deposit, and most likely fatty degeneration also, had been, for over a year, subject to attacks of angina pectoris which recurred at intervals of from one to three weeks, and always after sleeping. Her Allopathic physician had informed her friends that she would be likely to die in one of these attacks, and that he expected to do the same, as he was afflicted in the same way. The latter part of the prophecy was shortly afterwards fulfilled, and after his death I was

called in to treat his patient. Up to this time she had been steadily growing worse. After a careful survey of the case, I decided that Lachesis was the proper remedy, as indicated by her age, the change through which she was passing, the hot flashes, and the symptom "aggravation after sleeping." I gave it in the thirtieth attenuation, with the effect of greatly improving her general health, and so far removing the angina that when last heard from, about a year later, she had not had a paroxysm since she began taking my medicine.



## CHAPTER XIV.

### DISEASES OF THE AORTA.

In consequence of the relation which they bear to cardiac diseases, this work would not be complete without devoting some attention to atheroma, and aortic aneurism.

*Atheroma* has its original seat beneath the tunica-intima of the blood vessels, and consists in an increase of connective tissue elements, which afterwards take on either fatty or calcareous degeneration. This new tissue may be nearly uniform in its distribution, or in irregular patches or rice like bodies.

If the degeneration assumes the fatty form, the growth may be washed away, forming an ulcer, and so weaken the wall at that point that aneurism may result. When calcareous, the part has a stony feel, and the patches often form bony or chalk-like scales; or if the deposit be evenly distributed, the vessel may, in rare cases, be converted into a rigid tube. It is then said by some to be ossified, and becomes partially incapable of doing its share in carrying on the circulation. If a smaller artery be thus transformed, the blood supply may be entirely cut off from the part beyond, and gangrene ensue. Examples of this are sometimes met with in the feet of old people. If the aorta be the seat of such calcification, the coronary

arteries are, by reason of its loss of elasticity, no longer filled, and the heart suffers from diminished blood supply. The coronary arteries are themselves frequently the seat of disease, even in its worst forms, and dilatation and fatty degeneration of the heart thus hastened. The cerebral arteries, rendered brittle by the same process, thus become more liable to rupture, and apoplexy not unfrequently results.

Whether the degeneration be fatty or calcareous, one of the most constant results of the atheromatous process is dilatation of the vessel involved. If this be the aorta, it may generally be felt by pressing the finger well down in the sternal notch. The effect of such dilatation, as well as of the loss of elasticity, is imperfect recoil and deficiency of blood supply in the coronary arteries. The aortic orifice may participate in the dilatation, and the semilunar valves thus become inadequate to prevent regurgitation. The smaller arteries, when affected, are not only dilated but elongated and tortuous. The pulse then becomes both visible and unnaturally firm upon pressure.

The seat of the greatest atheromatous change is generally at those points at which there is the greatest strain upon the coats of the vessel, as on the outer side and ascending portion of the arch of the aorta, at bifurcations, and about the valves of the heart. The entire arterial system may be affected, and it may even extend into the veins at points where those vessels feel the force of the heart's contraction, either in

consequence of regurgitation or of communication with an artery.

Old age and the gouty diathesis are favorable but not necessary conditions for its development. Its cause is undoubtedly strain, as from hypertrophy or excited cardiac action, as already explained under the heads of hypertrophy and of aortic valvular disease. It is nearly always present in greater or less degree in Bright's disease.

The *prognosis* is certainly unfavorable, and no hope of a cure need be entertained, yet possibly the process may be arrested, and much may be done to prolong life. The patient often lives for many years after the atheromatous process has been established. Death usually takes place in consequence of heart failure.

The *treatment* should be directed, first, to the arrest of the atheromatous process; and second, to the removal of the consequences of the obstructed circulation.

For the first, such remedies as Arnica, Arsenicum, Lithium, Plumbum and Rhus tox. will be found most useful. Their indications may be found in the treatment of valvular disease.

For the second, see treatment of hypertrophy and dilatation.

Every thing which tends to increase the vascular excitement should be avoided. All kinds of spirituous liquors especially should be strictly prohibited. Tea and coffee are injurious.



## CHAPTER XVI.

### THORACIC ANEURISM.

From its close proximity to the heart, thoracic aneurism may be confounded with the affections to which that organ is subject. It will receive here a brief consideration.

Aneurism may be *true* or *false*. In *true* aneurism, all the coats of the vessels remain entire, but become dilated, rendering it considerably wider at the point affected. It is generally fusiform. When a considerable portion of the length of the artery is involved, it is merely said to be dilated.

In *false* aneurism, one or two of the coats, usually the middle and internal, have been broken through, leaving only the external, which forms a sac of varying dimensions upon one side of the vessel.

As varieties of the latter, are occasionally met with, *dissecting* aneurism, in which the blood, after breaking through the internal and middle coats, has formed a new channel for itself inside the external coat, and re-entered the vessel farther on; and *varicose* aneurism, when the aneurismal sac has formed a connection with some other vessel, as the pulmonary artery or a vein.

*True* aneurism is supposed to be connected with partial paresis of the vaso-motor nerve supply of the part. The direct or exciting cause of the lesion is

generally too great tension, either repeated or long continued, upon the walls of the vessel.

In *false* aneurism, it is not likely that the vessel has generally been weakened by disease previous to the rupture of the inner and middle coats, and the formation of the sac. The immediate cause of rupture may be mechanical violence, as a wound or some blow over the part, but is generally excessive vascular tension, either continuous or sudden. This increase of vascular tension is usually the result of some sudden and violent muscular effort.

Aneurism, then, occurs at the age when the greatest muscular power has been reached, four-fifths of the cases probably originating between the ages of thirty and fifty years. The majority of cases occur beyond the age of forty, the arterial coats becoming more brittle as age advances. Mechanics, laborers, porters, and others whose occupations call for violent muscular effort, are among its most frequent victims. For the same reason also, about seven-eighths of the cases are men.

The point of the most frequent appearance of aneurism is where there is the greatest vascular tension. Accordingly, more than one-half the cases have their seat in the ascending portion of the arch of the aorta, the frequency of its appearance rapidly diminishing as the distance from the heart increases.

The onset of the aneurism is often, but not usually, felt by the patient, as a sensation of something giving way during violent muscular effort; yet, for months

after this, he may feel no inconvenience from it, unless it be that his breath is somewhat shorter or he is more easily fatigued than formerly; but, sooner or later, the consequences must be serious. Once established, it must surely increase, every systole of the ventricle adding to its magnitude. Of course the more powerful the heart's action, the greater will be its effect upon the aneurism.

As the disease advances, the sac is apt to become irregular in outline, according to the space which it occupies. It presses upon neighboring organs, displacing them and interfering with their functions, and even eats its way by erosion, through the ribs or the bodies of the vertebræ.

One of its most constant symptoms is pain. This is of two kinds. The one is a constant dull aching, and has its seat in the aneurism itself; the other sharp, lancinating and intermitting, and resulting from the pressure upon adjacent organs. The pain of erosion of a bony substance is of a boring character, and when the spinal column is the point of attack, is always accompanied by tenderness of the corresponding vertebræ.

Other symptoms depend upon the extent and position of the aneurism, and the organ pressed upon. Pressure upon the œsophagus of course interferes with deglutition; or upon the laryngeal nerve, which curves round the arch, gives rise to laryngeal spasm, and to that peculiar ringing cough so characteristic of thoracic aneurism. Pressure upon the cervical sym-



pathetic ganglia occasionally produces dilatation of the pupil. Dyspnœa is present, not only in consequence of pressure upon the lung itself, but upon the trachea and bronchial tubes, and upon the pulmonary veins. Hæmoptysis is not uncommon. Pressure upon the inferior or superior vena cava, gives rise to venous congestion of the corresponding part.

The physical signs are not of much value unless the tumor comes in contact with the chest wall. Pulsation can then usually be seen and felt, and there will be dullness over the space corresponding to the area of contact. Pulsation and dullness resulting from aneurism will be found elsewhere than over any portion of the cardiac region, except in those rare cases in which the lesion has its seat in one of the sinuses of valsalva. In that case, diagnosis would be extremely difficult. Aneurism having its seat in the ascending or transverse portion of the arch, will make its appearance, if at all, near the sternum, and *above* the cardiac region. A perceptible tumor thus formed is distinguishable by the readiness with which it is reduced by compression with the hand. It is likely also to be more prominent during periods of cardiac excitement, or it may permanently disappear in consequence of having found additional space by displacing some other organ.

The impulse is generally single and systolic, but is occasionally double, being systolic and diastolic. It must not be confounded with the pulsation sometimes communicated to a solid tumor. Aneurism is some-

times accompanied by thrill from roughness of the orifice. Frequently, also, there are heard within the aneurism, sounds corresponding in point of time to the true heart-sounds. They are, however, generally somewhat louder, and the second is often booming in quality. They probably originate within the sac itself.

Thoracic aneurism may have many different terminations, according to its seat and the circumstances surrounding it. It may find its way to the surface, and the integument, becoming red and inflamed, slough away and produce instant death, or the opening may be closed by a clot of blood and the patient live for several days after the accident. It may break into the pericardium or pleural sac, producing speedy death, or into the spinal canal with like result. A slight blow over the part is often sufficient to produce rupture. In other cases they die of marasmus, or the patient is worn out by pain, dropsy and dyspnœa.

The *prognosis* is decidedly unfavorable, nearly all cases proving fatal; yet, with care, several years may elapse before the fatal end. Although occasional cures are made, spontaneous recovery is almost unknown.

The *treatment*, though still generally unsuccessful, is becoming with each decade more satisfactory. The object is two fold; to alleviate the sufferings of the patient, and to cure.

A cure is to be looked for, if at all, through solidification of the contents of the sac. Perhaps the best

method for bringing this about is what is known as Tutnell's method, which has for its object, absolute rest, and the greatest possible reduction of the volume of the blood without impairment of *quality*. The patient is strictly confined to the horizontal posture for a period of from two to three months, according to the progress of the cure. The bed must be so arranged that defecation and urination can be effected without rising, so that the horizontal position may be maintained without interruption. The patient may, however, turn slowly from one side to the other, or upon the back or face, always guarding against any sudden motion that might excite the heart to increased action, or disturb the process of coagulation within the sac. During this time the amount of food and drink taken must be *very limited*. For breakfast he may have an ounce of white bread and butter, with an ounce of milk; for dinner, three ounces of meat with three ounces of bread and potatoes, and four ounces of water or milk; for supper, two ounces of bread and butter and two ounces of milk or tea; thus making the total amount of solid food taken in twenty-four hours, nine ounces, and of liquid food seven ounces. Of course this must be varied according to the size and requirements of the patient. If he grows restive under the restriction, it is better to increase the allowance a little than to have the circulation excited by worrying.

A method somewhat similar to this was formerly pursued, but the patients were reduced to the starvation point, and further weakened by the occasional



abstraction of blood. The result of such treatment could only be the hastening of the poor victims into the grave.

Coagulation by galvano-puncture is growing in favor, and already has many advocates, among the most prominent of whom is Ciniselli, who claims to have cured five out of twenty-three cases operated upon. One died after the operation, from gangrene at the seat of the puncture. Some cases have required but one operation, and others four or five. The number of operations required seems to depend not only upon the size of the sac, but of the opening into the artery as well.

After solidification of the contents of the sac, by whatever method, the tumor shrinks to a comparatively small size, with increasing relief to the patient.

As to the medicinal treatment, Old School physicians claim to have used the Iodide of Potassium with good results in a few instances. If this drug has really cured any cases, the secret of its beneficial effect most likely lay in its power to diminish the quantity of the blood, and thus diminish the tension. Ergotin and the Acetate of Lead have also found some favor with them. In most instances, however, they have contented themselves with purely palliative measures, relieving the pain as best they could by administration of Morphia, either by the mouth or hypodermically, and diminishing the tension by the abstraction of a few ounces of blood.

Our Homœopathic literature on this subject is

scarce. Whether pursuing the method of Tufnell or that by galvano-puncture, such medicines may be added as seem Homœopathic to the case, both to the end of affording relief, and of curing the aneurism.

It is a well known fact that painters, more than others, are subject to aneurism, and Homœopathic physicians might take a hint from that, yet I would not advise the use of Plumbum without absolute rest, and at least a quite limited diet, for it would be almost certain to be attended with failure.

Dr. Richard Hughes reports a cure of a case of carotid aneurism in four days by *Lycopodium* 12th. I can only say that in my opinion this must have been one in which there was no rupture of the coats of the vessel, but merely a state of local dilatation, (true aneurism), and that it must have been of moderate degree and of recent origin.

“Best remedies so far as known: 1. *Carbo. veg.*, *Lach.*, *Lyc.*; 2, *Guai.*, *Puls.*, *Sulph.* In some cases may be required, 3, *Calc.*, *Caust.*, *Graph.*, *Kali*; 4, *Ambr.*, *Arn.*, *Ars.*, *Aur. m.*, *Fer.*, *Natr. m.*, *Zinc.*” (*Lilienthal.*) To these I would add *Plumbum*.

Among those best suited to control the cardiac irritability, and thus in a measure relieve the tension, are *Aconite*, *Bryonia*, *Cactus*, *Digitalis*, (not lower than the 3rd), and *Veratrum vir.*

For the relief of the severe pain, such remedies as *Arnica*, *Cactus*, *Hypericum*, *Rhus tox.* and *Spigelia*, are likely to be found most useful.

The application of cold should not be forgotten. It

should not, however, be so long continued as to endanger the vitality of the part, as fatal sloughing may ensue.

The extrinsic symptoms are so various, according to the organs encroached upon, that it would be impossible to prescribe any treatment for them in advance. Most of these will be more or less relieved by reducing the tension upon the aneurism.

In all cases, our endeavor should be to relieve as far as is in our power, and to postpone the fatal end. When not pursuing any such method as that of Tuffnell above described, he should have a liberal and nutritious diet, in order to preserve his strength and the tone of the arterial coats. All blows, all violent exertion, or shocks of any kind, and all cardiac excitement, should be strictly guarded against.



## CHAPTER XVII.

### CONGENITAL DEFECTS.

These usually consist of such a malformation of the heart or great vessels as to interfere with the normal circulation of the blood. Just how far intra-uterine inflammation is responsible for the occurrence of these phenomena, it is difficult to say.

With those cases in which there is no heart, a simple tube taking its place, we have here nothing to do. The heart may, however, be misplaced, either within the thorax, as when it occupies the right side of the chest entirely, in which case the liver and spleen generally also exchange places; or outside of it, as when it is found in the cervical region or in the abdomen. Flint mentions a case in which it occupied the place of the right kidney. In a case which came under the observation of the writer some years ago, the heart was found occupying the centre of the left half of the thorax, the sternum being so much depressed as to render the normal position impossible.

The *pericardium* may be partially or entirely wanting, yet most of the supposed cases have probably been erroneously so reported, the membrane having merely become uniformly adherent.

The most frequent malformation of the heart itself, is *stenosis* of the orifice of the *pulmonary artery*, with

or without atresia of that vessel. It is no doubt, in most cases at least, the result of endocardial inflammation. The stenosis may be effected by contraction of the tissues surrounding the orifice, by the edges of the valves adhering together, or by both of these processes combined. Perhaps most of those cases in which the valves are said to be deficient in number may be best explained in this way; two or more of the segments having been thus united into one.

All other things being equal, it may be said that the earlier the stenosis occurs, the smaller and more delicate will be the pulmonary artery. When that vessel is nearly or quite normal, the only obstruction to the pulmonary circulation being at the constricted orifice, a murmur is likely to be heard in the left second intercostal space, close to the sternum. It is systolic in time and not transmitted. Blood passing through the septa from one auricle or ventricle to the other, seldom if ever gives rise to any murmur.

Another congenital defect not so frequently met with, is *stenosis* of the *conus arteriosus*. This may be continuous with pulmonary stenosis, in which case the entire conus may be converted into a more or less rigid tube, or the constriction may be confined to a single portion at some distance from the pulmonary opening, and the part so affected be converted into a narrow neck, thus cutting off the conus from the main body of the ventricle, with which it communicates by a narrow opening. In this case, the part so separated is by some considered as an additional ventricle.

Stenosis of the conus as well as of the pulmonary orifice, is of course liable to interfere more or less with the pulmonary circulation. In either case, either the foramen ovale or the inter-ventricular septum, or both, will usually be found open, a portion of the venous blood, more or less mixed with the arterial, going to the lungs through the ascending aorta, the ductus arteriosus, and the pulmonary branches. Should the pulmonary artery and the ductus arteriosus both be closed, the deficiency may be partially compensated by the bronchial arteries.

The *foramen ovale* may remain open after birth in consequence of a disproportion between the size of the valve and of the orifice to be closed. More frequently, however, it is a case of simple non-adhesion, a portion of the venous blood rushing through from the right auricle to the left with each auricular systole. How large a portion of the cases of open foramen ovale after birth are dependent upon obstruction to the pulmonary circulation is a mere matter of conjecture.

The inter-ventricle septum may be wide open, in which case the obstruction to the pulmonary circulation must have taken place very early in foetal life, the current passing from the right side to the left having prevented the formation of a proper septum. In other cases it may merely present an opening at the membranous or undefended portion at the base of the ventricle, or a small portion of the muscular substance may also be wanting.

The left ventricle is likely to be somewhat smaller



and thinner than natural, while the right is considerably hypertrophied and generally exceeds the left in size, and in the thickness of its walls. This is to be expected, as a natural result of the extra amount of work devolving upon it, not only in forcing a small portion of the blood through the narrowed pulmonary orifice, but in doing a large share of carrying on the systemic circulation as well; yet, in rare cases, the reverse is true, the right ventricle being little more than rudimentary. Here the venous blood seems to pass almost directly from the right auricle into the left ventricle, compelling it to do double duty.

To distinguish between congenital narrowing of the pulmonic orifice, and that which has been acquired after birth is not always easy. According to Kussmaul, it is more certainly congenital: "First, when the birth was near the normal end of pregnancy; second, the sooner after birth cyanosis and other tokens of heart disease, collectively called physical symptoms of stenosis of the pulmonary artery, are perceived; third, when the foramen ovale and the ductus arteriosus botalli are both open, or, indeed, only the latter; fourth, when the opening of the foramen ovale is proportionately large, the ductus being closed, and especially when its size depends on want of the fleshy substance of the septum; fifth, when the valves of the pulmonary artery show anomalies of structure that are evidently congenital; sixth, when the trunk of the pulmonary artery is decidedly contracted and its walls too thin; seventh, when the right ventricle appears con

tracted, or stunted." Of these it is evident that only the first two can be made available as a means of diagnosis during the life of the patient, the second being much more important than the first.

Of the symptoms and physical signs, there is not one which can be properly said to be pathognomonic; yet some of them are highly suggestive, and, when they are taken together, an approximate diagnosis should not be attended with great difficulty. The enlarged right ventricle may generally be seen and felt, beating with more than usual force, in the space from the apex to the sternum, and the area of dullness may be increased somewhat both to the right and left. In addition to this, a systolic murmur may not unfrequently be heard in the second left intercostal space, close to the sternum. These patients are, from the beginning, usually cyanotic, and suffer more or less from shortness of breath. They have cold extremities, are easily chilled, are troubled with dizziness and headache, and show a general lack of bodily vigor. These symptoms may, however, not manifest themselves immediately after birth; or, if present, they may gradually diminish in intensity, especially if the collateral circulation is pretty good. Some even go on to adult age with but little if any inconvenience. In such cases, the symptoms of heart failure may set in suddenly, as after some sudden shock or a severe illness, but more frequently they are very gradual in their development.

The cyanosis of infants thus affected may be distin-

guished from that often seen as a consequence of partial strangulation during labor, by the fact that the latter passes quickly away, while the former is persistent, although liable to exacerbation under certain circumstances, as from laughing, crying, or changing position. It is usually more marked than that resulting from heart failure dependent upon congenital diseases. Various opinions have been expressed as to its exact cause, some claiming that it is dependent upon the admixture of the venous blood with the arterial, while others maintain with equal earnestness that it is due to stagnation of blood in the veins, and especially in the venous capillaries. For my own part, I see no reason for adopting either of these views to the exclusion of the other, and, indeed, it seems to me that the imperfect aeration of the blood, must, in many cases at least, play a very important part in its production.

*Terminations.*—Children in whom there is scarcely any collateral circulation, grow weaker, the skin becomes cold, and they die either asphyxiated or in a slow collapse. (Lebert.) Those who survive for a considerable length of time, afterwards die with all the symptoms of heart failure from other causes, as bronchitis, cardiac asthma, cyanosis and general dropsy. Lebert notices a very marked tendency to tuberculosis, in contrast with the immunity of those suffering from left side disease.

*Prognosis.*—This of course depends upon the degree of obstruction to the flow of blood through the pul-



monary artery, and the efficiency of the collateral circulation. According to Hayden, those suffering from atresia seldom live even a few days. Kussmaul makes a better showing, fourteen out of twenty-five dying during the first year, while one, (Voss), lived thirty-seven years, but in this the collateral circulation was unusually good. The prognosis of stenosis of the pulmonary artery or of the conus, is of course much more favorable, yet of these, forty-one of the sixty-four cases collated by the same author, died before the tenth year.

*Treatment.*—Of course no real cure is to be thought of. The best that can be done is to secure as high a degree of bodily vigor as possible, both by the strict observance of hygienic rules, and the timely administration of the proper Homœopathic remedy when needed. The medicine most likely to be required will be found under the head of hypertrophy and dilatation.

## CHAPTER XVIII.

CAUSES OF DISEASES OF THE HEART.—DISTINCTION  
BETWEEN CARDIAC AND NON-CARDIAC AFFECTIONS.

—ELEMENTS OF PROGNOSIS.

Hayden, in his work, classifies the causes of cardiac disease into hæmic, mechanical, and nutritive.

Hæmic causes are such as operate through the presence of certain irritant principles in the blood. As examples of these, are found:

Rheumatism.	Small-pox.	Scurvy.
Scarlatina.	Septicæmia.	Purpura.
Chronic renal dis-	Puerperal fever.	Cancer.
ease.	Phlebitis.	Syphilis, and tu-
Measles.		berculosis.

Most prominent among them, and indeed the most prolific of all causes of cardiac disease, is acute rheumatism. The resulting complication is either pericarditis, endocarditis, or both combined. Their relative frequency has already been discussed in a previous chapter. The cardiac inflammation may now and then be the first manifestation of the rheumatic attack, but usually the heart does not become involved until several days have elapsed from the beginning of the disease. Various estimates have been made as to the percentage of cases of acute and sub-acute rheumatism

in which the heart is involved, each author drawing mostly from his own experience. In Latham's cases the complication was present in two-thirds; in Sibson's and Hayden's, something less than one-half. Fuller's tables show a proportion of something over one-third. In my own private practice the percentage has been very much less.

Although the contrary has frequently been asserted, my own experience leads me to believe, with Vogel, Rosenstein and others, that the severity of the rheumatic inflammation has little if anything to do with the liability to cardiac complication. Much more likely is it to depend upon the number of joints affected at the same time, and the rapidity with which the inflammation moves from one part to another. No doubt much depends upon the treatment. If the rheumatic attack can be cut short in the first few days, the heart is not so likely to participate.

Age seems to exert a great influence, the younger the subject of acute or sub-acute rheumatism, the greater being the liability to cardiac complication. Hayden expresses the opinion that it is only exceptionally absent in those children who are attacked before the age of twelve years. In old persons its occurrence is rare.

Perhaps the poison of scarlatina should be classed next to that of rheumatism as a cause of cardiac disease. The form of the resulting complication is usually endocarditis or pericarditis. It generally appears during the decline of the primary affection. The endo-



carditis, like that of rheumatic origin, usually attacks the mitral valve. The exudation into the pericardium, when that structure is the seat of the inflammation, is generally serous.

Endocarditis may likewise result from the poison of measles or small-pox, while in the typhoid form of small-pox, as well as in phlebitis and puerperal fever, we are more likely to have pericarditis, which is not usually recognizable except by its physical signs. (Hayden.)

Purpura and scurvy may now and then be complicated by pericarditis of a hæmorrhagic form.

Pericarditis may also result from cancer and from tuberculosis.

Another prolific cause of disease of the heart is Bright's disease of the kidney. The form of the resulting complication is generally hypertrophy of the left ventricle. This is present in a very large proportion of those cases which terminate fatally in the third stage of the disease. The aortic valves are also affected in many cases. The retained urinary excreta excite spasm, and finally thickening of the coats of the minute systemic arteries, and this in turn acts as a mechanical cause of hypertrophy, by offering resistance to the circulation. Dr. George Johnson has summed it up thus; "Blood contamination by retained excreta, capillary resistance, hypertrophy of the minute arteries, and hypertrophy of the left ventricle." The aortic valvular affection is an after effect, and results from the powerful recoil of an aorta which has been over

distended by the contraction of a hypertrophied left ventricle.

Among the most important of the mechanical causes is constriction of any part of the body, but especially the waist and chest, as by belts, corsets, or other close fitting apparel; but when violent exercise is added to these, the practice becomes doubly injurious. The usual result is hypertrophy of one or both ventricles, to be followed afterwards by dilatation. In those who are but poorly nourished, dilatation may predominate from the beginning.

Violent labor in a constrained position, as in strikers at the anvil and miners in thin seams of coal will be likely to produce more or less hypertrophy of the left ventricle.

Swimming and other sports which call for violent and rapid respiration are likely to produce enlargement of the right ventricle.

Nearly all the mechanical causes so far enumerated, not only contribute to the production, either primarily or secondarily, of disease in the aortic valves, but are also prolific causes of thoracic aneurism.

Such a deformity of the chest as to interfere with the heart's action may be a cause of hypertrophy of the left ventricle; or the heart may be so placed as to form an angle in the aorta, which would prove an obstruction to the circulation, and might be a cause of hypertrophy.

Anything which interferes with the pulmonary circulation, as a tumor pressing upon the pulmonary

artery, long standing disease in the lungs, as emphysema, tubercular deposits, hepatization, and pleuritic effusion, may be a cause of enlargement of the right ventricle.

Wounds of the heart or pericardium are not likely to give rise to long standing disease, as they usually terminate in either speedy death or recovery. Falls or blows upon the chest may produce rupture of the chordæ tendineæ, or rupture and detachment of the semi-lunar valves.

Repeated and long lasting exposure to cold may be a cause of hypertrophy, by obstructing the circulation of blood in the capillaries.

Habitual excitement of the heart, as by the use of spirituous liquors, is also a frequent cause.

High living in general, by producing a gouty condition, with atheromatous arteries, may be a remote cause of imperfect coronary circulation, fatty degeneration, and angina pectoris.

Tobacco smoking causes cardiac debility with distressing palpitation, and frequently also a slight degree of dilatation.

Imperfect nutrition may lead to atrophy, softening, or fatty degeneration.

*Distinction between Cardiac and Non-Cardiac Affections.*—The apparent similarity between certain symptoms and diseases which are not of cardiac origin, and certain other affections which are cardiac, not unfrequently leads to confusion. Perhaps one of the most frequent errors of this kind is to mistake left side pleur-



itis for pericarditis, and *vice versa*. If a friction sound is present, there is likely to be little if any difficulty as long as we remember that in pericarditis this is of cardiac rhythm, and heard only in the precordial region, while that of pleuritis is respiratory in rhythm, is heard rather over the lungs than the heart, and ceases on holding the breath. In those rare cases in which there is pleuritic murmur of cardiac rhythm, resulting from inflammation of that portion of the pleura which is reflected on the external surface of the pericardium, it is not heard in the precordial region, as in pericarditis, but rather at the left hand border of it.

Extensive *effusion* within the *pericardium* may be distinguished from *pleuritic effusion* by the percussion dullness, which in the one case is precordial and somewhat wedge-shaped, while in the other it is over the lung of the affected side, and always seeks the lowest point. In extensive pericardial effusion, also, the apex beat is imperceptible when lying upon the back, but again becomes perceptible when assuming the prone position, and usually also when lying upon the left side.

Simple *bronchitis* may be distinguished from that which results from failure of the mitral valve, by the systolic apex murmur in the latter; also by the dyspnoea when lying down, the palpitation on exertion, the irregular pulse, and, after the tricuspid gives way, general dropsy.

*Hæmoptysis*, either vicarious or resulting from pulmonary disease, may be distinguished from that which

results from stenosis of the mitral orifice, by the absence of the presystolic apex murmur which is nearly always present in the latter.

The *bellows murmur* of *anæmia* might be confounded with organic endocardial murmurs. It may be readily distinguished from mitral murmurs, by always having its point of maximum intensity at the points of origin of the aorta and pulmonary artery respectively, instead of at the apex. Like the murmur of aortic stenosis, it is basic and systolic, but it may be distinguished from that, by its soft and blowing character, and by not being transmitted along the course of the aorta, and into the carotids, as is the obstructive murmur. The anæmic murmur also often becomes inaudible on assuming the recumbent posture, and is more frequently met with in women and in persons who are generally thin and bloodless.

*Hysterical asthma* very closely resembles angina pectoris, about the only distinguishing feature being the absence of pain in the former.

*Palpitation of reflex* origin, as that which results from gastric disorders and from disease of the uterus and ovaries, sometimes leads to confusion, but differs from that of heart failure by being worse during rest, while the latter is aggravated by exertion. The palpitation and irregular and excited action of the heart, met with in the so-called spinal irritation, although in one sense made worse by exertion, still differ from the same symptoms resulting from heart failure in this; that the former are especially aggravated by

fatigue, while the latter are worse upon sudden, violent exertion.

Both forms may be further distinguished from that resulting from heart failure, by the absence of organic murmur, increased area of dullness, or other physical signs of organic disease.

The *excited action* of the heart met with in chronic pulmonary disease is rendered more apparent by the thinness of the chest walls. It should not be mistaken for dilatation, as there is no increase in the area of dullness, and the apex is found beating in the normal position.

*Pain* in the region of the heart very often occurs as a result of gastric or other disorders, but the physical signs of organic heart disease are absent.

*Elements of Prognosis.*—The most important element in the prognosis of cardiac affections, is the degree of power possessed by the heart muscle, as evinced by the force of its contraction. As long as these continue to be energetic, and without excitement, the prognosis, as to time, may generally be considered good, but when hypertrophy gives way in dilatation, as shown by the increasing breadth of dullness, and the more diffused and feeble slap with each ventricular systole, instead of the more concentrated and powerful apex beat of pure hypertrophy; or later, when the impulse grows very feeble or perhaps imperceptible, no serous effusion or excessive adipose being present to obscure it, and especially if associated with these symptoms, the first sound is either faint or inaudible,



our gravest fears might well be excited. Other evidences of the loss of power are intermittency, irregularity in rhythm, and palpitation, especially if made worse upon exertion. Intermittency upon exertion is especially indicative of fatty degeneration.

Concerning the prognosis of left side valvular disease, perhaps that of aortic regurgitation is most unfavorable, the mitral lesions coming next to this, while aortic obstruction is least dangerous, but in none of these, with the exception of mitral stenosis, is there usually much danger of speedy death until the signs of failure of the left ventricle have become manifest. Occasionally, however, in the case of aortic regurgitation, or even of moderate obstruction, if combined with renal disease, we have sudden death by apoplexy, from the rupture of an atheriomatous artery by the too vigorous contraction of a powerful left ventricle.

Generally speaking, as long as the compensation continues good, the patient can go about his usual occupation, and may live for many years in tolerable health. In mitral narrowing, of course it is the left auricle which first fails, and afterwards the right ventricle. When, in any case, failure of the right ventricle has gone on to the extent of rendering the tricuspid incompetent, as shown by the pulsation in the veins, the general venous engorgement, cyanosis, and œdema which begins in the feet and extends upwards, a period of a few weeks is generally sufficient to end the scene.

Yet, by remedies calculated to increase the general

vigor and strengthen the heart walls, even this dropsy may be removed for a time, only to return with increased intensity. Dropsy of that form which results from combined heart and kidney disease, and which more frequently begins about the eyes, or perhaps the genitals, is usually more unsteady in character, and hence not so indicative of speedy death. It may appear and disappear many times in the course of the disease, each return being worse than the one which preceded it.

*All mitral systolic murmurs* are the result of *mitral regurgitation*, but *not* all are the consequence of *disease* of the mitral valve. This valve is not rarely rendered incompetent by the giving way of the ventricular wall at the origin of a papillary muscle during systole. This form of mitral incompetency is most frequently met with in pericarditis, myocarditis, typhoid and typhus fevers, and chorea. Regurgitant murmur thus produced, is not so constant as that which results from valvular disease, and is capable of removal by any remedy which strengthens the heart muscle, or corrects the faulty innervation.

Regurgitation, the result of valvular disease, is seldom if ever cured, yet under certain circumstances, the *murmur* of regurgitations resulting from disease of the mitral valve is liable to cease, the regurgitation still persisting. These conditions are supposed to be the failure of the tricuspid valve, and great debility of the left ventricle. In the latter condition, the murmur is sometimes temporarily restored by increasing the ventricular power, but in the last stages,

after the vitality of the patient is nearly gone, such restoration is impossible. From this, it will be seen that the cessation of a mitral regurgitant murmur resulting from disease of the valve, is of unfavorable augury.

While the cessation of the pulmonary symptoms in mitral disease may be the result of increased ventricular power, and hence a favorable sign, yet we should not fail to look upon it with some degree of suspicion, since it very commonly follows closely upon the failure of the tricuspid.

Loss of arterial tension is generally shown by the diminution in the amount of urine secreted, and should be closely watched.

General venous engorgement, as shown by dropsy, cyanosis, and congestion and impairment of function of the various organs, will be found to exist in an inverse proportion to the degree of arterial tension, and especially is this true after the failure of both the mitral and tricuspid valves.

Both loss of arterial tension, and general venous engorgement, result from the same cause, viz: valvular incompetency and failing ventricular power. Anything, therefore, which increases the power of the heart and the energy of its contractions, increases the amount of urine. When, along with tricuspid failure, this is almost entirely suppressed, and especially if albumen and tube casts can be detected, the outlook is indeed most serious.

As to sudden death, it is now time that not only



physicians but laymen should divest themselves of the idea that every form of cardiac disease is liable to such termination. In fact, there are only three forms of disease of the heart in which such a result is to be feared. These are: first, hypertrophy of the left ventricle with atheromatous arteries, as in aortic disease; second, fatty degeneration; third, angina pectoris. Even in these cases the decline is much more likely to be gradual. This constant dread of sudden death is not only a source of great suffering and anxiety to many, but may hasten the fatal end when the cardiac lesion might otherwise remain harmless for many years. Perhaps nearly every old practitioner can call to mind some one who has had his life poisoned and rendered worse than useless, by the incautious use of the dreaded expression "heart disease," when if he had been suffered to remain in ignorance, he might, for a considerable time at least, have led a life of usefulness, both to himself and to others. While I would not be understood as encouraging deception in the main, yet it is not always best to announce to the patient himself, the discovery that he has a disease of the heart, particularly if he is yet in tolerable health. In speaking to him of his troubles, it would be better, if the cardiac affection is mentioned at all, to allude to it only vaguely, or as if it were only a symptom.

Concerning the *treatment* of cardiac disease in general, a few words in closing might not be amiss. In treating the acute inflammations, although of course

having a care to the maintenance of the vitality of the patient, our remedies must in the main be directed to the inflamed part. On the other hand, in treating the more chronic affections, as chronic valvular disease, dilatation, etc., where heart failure is the one thing to be guarded against more than all else, much more can be accomplished by directing our attention to the general vitality, and especially securing as healthy a condition of the digestive organs as possible, at the same time, however, not neglecting such remedies as have a special Homœopathic relation to the particular form of cardiac affection present. Above all, the physician should not suffer himself to fall into the lazy habit of merely generalizing. Each case should be studied closely, and the remedy selected in accordance with the totality of the symptoms, giving due prominence to characteristics.

# INDEX.

## A

Adhesion of pericardium, 70.  
 Anatomy of heart, 17.  
 Aneurism, cardiac, 159, thoracic, 240.  
 Angina pectoris, 225, 144, distinguished from cardiac asthma, 227; treatment, 228; cases, 234.  
 Aorta, diseases of, 237.  
 Aortic opening, 21; recoil, 25.  
 Apex, position, 31; displacement, 37.  
 Appendages, auricular, 20.  
 Arteries, tortuous, etc. 39.  
 Aspiration, 81.  
 Asthma, cardiac, 227.  
 Atheroma, 237.  
 Atrophy, cardiac, 128.  
 Auriculo-ventricular opening, left, 21; right, 20.

## B

Basedow's disease, see exophthalmic goitre.  
 Bellows murmur of anæmia, 262.  
 Blood, supply of to heart walls, 24.  
 Bright's disease, 83, 91, 94, 258.

## C

Cancer of heart, 158.

Cardiac and non-cardiac diseases, distinction between, 260.  
 Causes of cardiac diseases, 256.  
 Cheyne, symptom of, 158.  
 Chordæ tendineæ, 22.  
 Circulation of blood, 18.  
 Columnæ carneæ, 22.  
 Congenital defects, 249.  
 Consequences of hypertrophy, 106.  
 Conus arteriosus, 20, 250.

## D

Digitalis, action of, 118.  
 Dilatation, 108; physical signs of, 109.  
 Doubling of sounds of heart, 43.  
 Dullness, deep, normal area of, 33; superficial, 32.  
 Dullness, increased area of, 42.

## E

Embolism, 207.  
 Endocardium, 23.  
 Endocarditis, 163; etiology, 163; history, 164; symptoms, 167; physical signs, 168; ulcerative, 170; prognosis, 171; chronic, 172; treatment, 173.  
 Exaggeration of heart sounds, 43.



Examination of heart, physical 29.

Exophthalmic goitre, 219.

## F

False hypertrophy, 127; etiology, 128.

Fatty degeneration, 134; conditions, 136; causes, 137; physical signs, 138; symptoms, 142; angina pectoris as symptom, 144; history, 145; prognosis, 147; treatment, 147.

Fatty infiltration, 153; diagnosis, 154; prognosis and treatment, 155.

Friction sounds, pericardial, 49; pleuritic, 50.

Fremissement cataire, 38.

## G

Ganglia of heart, 26.

Granular softening, 156.

## H

Hydatids, 159.

Hydro-pericardium, 83; prognosis, 84; treatment, 84.

Hypertrophy, 88; classification, 90; causes, 91; obstructed circulation, 91; distention under increased pressure, 95; habitual excitement of heart, 97; left ventricle, 99; pure hypertrophy, 100; excentric, 102; auricles, 103; right side, 103; physical signs, 104; consequences, 106; dilatation 108; physical signs of, 109; prognosis of cardiac

enlargement in general, 111; treatment, 113; false hypertrophy, 127.

## I

Inhibitory nerves, 27.

Innervation, cardiac, 26.

Intermittency, 40.

Irritable heart, 216, treatment, 217.

## M

Murmurs, 44; table of comparison, 47; classification, 47; combinations, 47; hæmic, 48; dynamic, 48; venous, 48; pericardial, 49.

Myocarditis, 131; etiology, 132; diagnosis, 132; prognosis, 133; treatment, 133.

## O

Obstructed circulation, effects of, 51; brain, 53; liver, 54; kidney, 54; œdema, 55; pneumonia, 57; prognosis, 57.

## P

Palpitation, 41, 209; prognosis, 210; treatment, 210.

Pause, long, 34; short, 34.

Pericarditis, 58; physical signs, 62; pericardial and endocardial murmurs compared, 67; terminations, 69; recurring, 72; chronic, 72; complications, 72; prognosis, 73; treatment, 74; hygiene, 82.

Pericardium, 23.  
 Physical signs of heart in health, 31.  
 Physical signs of cardiac disease, 37.  
 Physiology of heart, 26.  
 Pneumogastric nerve, 26, 27.  
 Pneumopericardium, 86; treatment, 87.  
 Prognosis, elements of, 263.  
 Pulmonary artery, 20.  
 Pulse, 39.

**R**

Recoil, aortic, 25.  
 Rest or sleep of heart, 27.  
 Retraction, systolic, of ensiform cartilage, 37.  
 Rhythm of heart sounds, irregularity in, 41.

**S**

Semilunar valves, 21, 22.  
 Simple softening of heart, 156.  
 Sounds of heart, time of, 34; location, 35; suppression, 43.  
 Spaces, bulging of intercostal, 37.  
 Sphygmograph, 35.  
 Stethoscope, 30.

Sudden death, 266.  
 Suppression of heart sounds, 43.  
 Syphilitic nodules, 158.

**T**

Tricuspid valve, 21; failure, 198, 266.  
 Tufnell's method, 245.  
 Tubercles, 159.

**V**

Valsalva, sinuses of, 23.  
 Valvular diseases, 179; classification, 180; mitral obstruction, 181; mitral insufficiency, 187; aortic obstruction, 192; aortic insufficiency, 197; right side, 198; tricuspid, 198; pulmonic, 200; prognosis, 201; treatment, 202; puncture of skin in, 205; purgatives in, 206; hygiene, 206.  
 Venæ Thebesii, 20, 25.  
 Ventricle, left, 21; right, 20.

**W**

Weight of heart, normal, 19.

## ERRATA.

Page 17, and after, head-lines; for "Physiological Anatomy" read "Anatomy and Physiology."

Page 41, 7th line; for "off" read "of."

Page 54, 1st line; for "the" read "this."

Page 55, 4th line; for "heart" read "heart's."

Pages 83 and 85, head line; read "Hydropericardium."

Page 109, 6th line from bottom; for "more" read "mere."

Page 110, 7th line; for "much" read "often."

Page 117, 6th line; for "patients" read "patient's."

Page 118, 3rd line; for "to" read "too."

Page 135, 5th line from bottom; for "III" read "IV."

Page 163, 11th line; for "disease" read "diseases."

Page 209 and after pages; omit "Neuroses" from head-lines.

Page 241, 3rd line; for "not" read "most."

Page 251, 21st line; read "inter-ventricular."

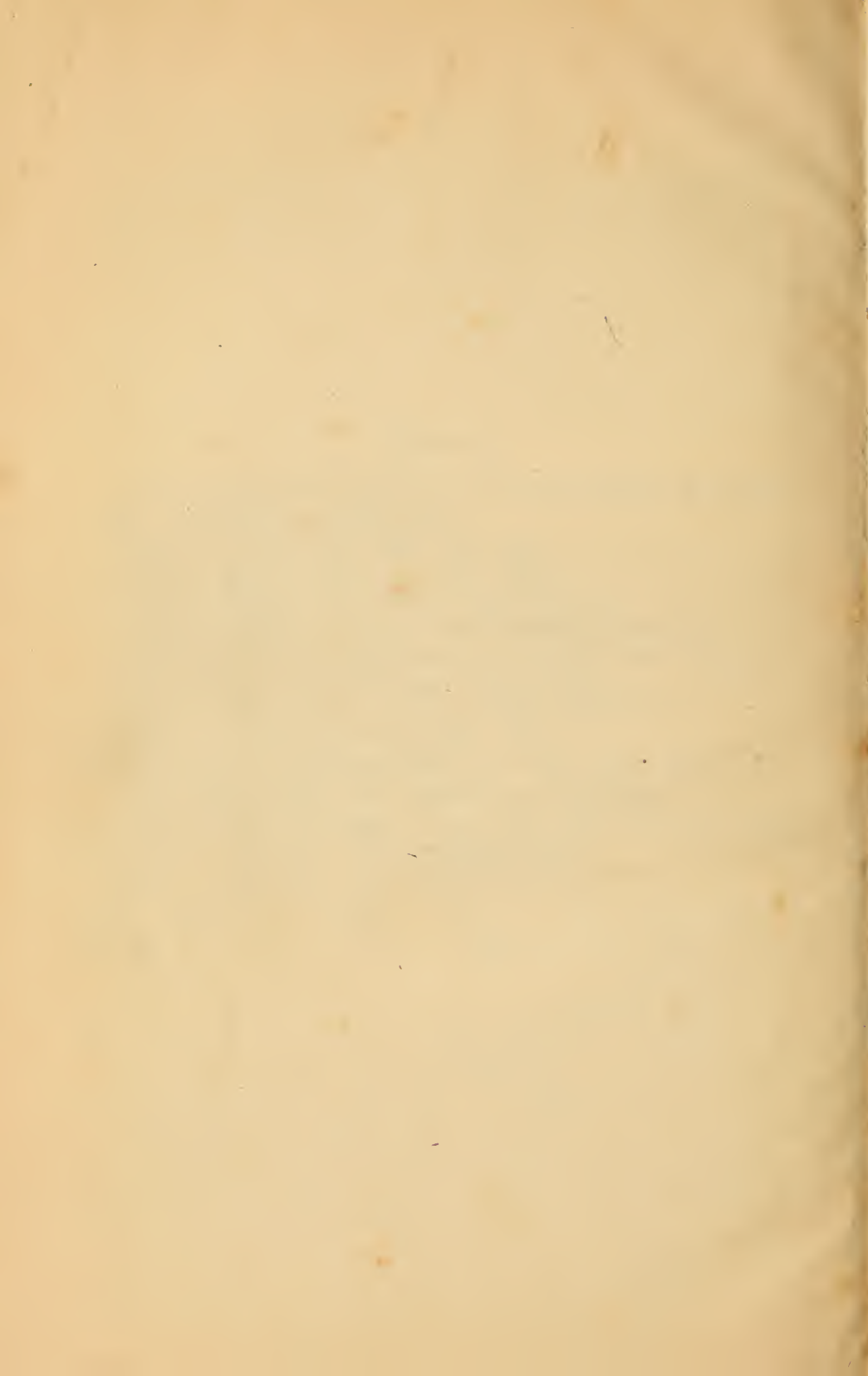
Page 251

4

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